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W. R. BIRDSALL, M.D., C. L. DANA, M.D., A. McL. HAMILTON, M.D., WM. A.  
HAMMOND, M.D., G. W. JACOBY, M.D., GRACE PECKHAM, M.D., E. C. SEGUIN,  
M.D., E. C. SPITZKA, M.D., M. ALLEN STARR, M.D., New York; C. F.  
MACDONALD, M.D., Auburn, New York; MORTON PRINCE, M.D.,  
Boston; L. C. GRAY, M.D., Brooklyn; J. G. KIERNAN, M.D.,  
Chicago; ISAAC OTT, M.D., Easton, Pa.; C. K.  
MILLS, M.D., Philadelphia; Prof. BURT G.  
WILDER, M.D., Ithaca, and others.

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VOLUME XIV

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ALTHAUS, JULIUS, M.D., M.R.C.P., London.  
BIRDSALL, W. R., M.D., New York.  
BROWNING, WM., M.D., Brooklyn.  
COLLMAR, CHARLES, M.D.  
DE SCHWEINITZ, GEO. E., M.D., Philadelphia.  
DA COSTA, J. CHALMERS, M.D., Philadelphia.  
DERCUM, FRANCIS X., M.D., Philadelphia.  
EDINGER, LUDWIG, M.D., Frankfort-on-the-Main.  
ESKRIDGE, J. T., M.D., Colorado Springs.  
VAN GIESON, IRA, M.D., New York.  
HOLT, L. EMMETT, M.D., New York.  
JACOBI, MARY P., M.D., New York.  
JACOBY, GEO. W. M.D., New York.  
KNAPP, P. C., M.D., Boston.  
LEIDY, PHILIP, M.D., Philadelphia.  
LLOYD, J. HENDRIE, Philadelphia.  
LYMAN, HENRY M., M.D., Chicago.  
MACDONALD, C. F., M.D., Auburn, N. Y.  
MASON, L. D., M.D., Brooklyn.  
MEIGS, ARTHUR V., M.D., Philadelphia.  
MILLS, C. K., M.D., Philadelphia.  
MITTENDORF, W. F., M.D., New York.  
OSLER, WM., M.D., Philadelphia.  
OTT, ISAAC, M.D., Easton, Pa.  
PARSONS, R. L., M.D., Sing Sing, N. Y.  
PORTER, WILLIAM H., M.D., New York.  
PUTNAM, J. J., M.D., Boston.  
ROBINSON, WM. DUFFIELD, M.D., Philadelphia.  
ROSSE, IRVING C., Washington, D. C.  
SACHS, B., New York.  
SEGUIN, E. C., M.D., New York.  
STARR, M. ALLEN, M.D., Ph.D., New York.  
WALLACE, W. H., M.D., Philadelphia.



THE  
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Original Articles.

REPORT OF A CASE OF BRAIN AND SPINAL  
CORD SYPHILIS, SHOWING EXTENSIVE  
DISEASE OF THE BLOOD-VESSELS.

By ARTHUR V. MEIGS, M.D.

WITH AN ACCOUNT OF THE AUTOPSY AND  
MICROSCOPIC EXAMINATION.

By GEORGE E. DESCHWEINITZ, M.D.

A MAN, then 63 years of age, had in January or February, 1877, a macular syphilitic eruption upon the face, trunk, and limbs, and, at the same time, a violently painful iritis. From these disorders he recovered, but continued in rather an ailing condition until September 13th, when, after having dragged the right leg, and having been somewhat awkward in walking for about two weeks, he was seized with right hemiplegia. The paralysis was not complete, although very pronounced, for he was still able to walk a little; there was also decided thickness of speech, although at this time there was no absolute loss of power of speech. After this attack, he slowly improved, and was able to be up until about December 1st, when, after suffering with some dimness of vision and feeling of general *malaise* and staggering gait, he was seized with violent vomiting and a fresh

access of paralysis, which was now almost complete, so that for a time he was unable to walk at all. Again he improved, but on May 13th, 1878, had another return of paralysis, which was complete, and there was entire loss of speech, except that he could say "yes" and "no." In the latter part of April or early in May, 1880, he had some vague increase of paralysis, and on May 23d had an attack, which was so sudden that he nearly fell, and afterwards complained of a strange sensation upon the left side. Upon investigation, it was found that there was almost complete loss of cutaneous sensibility in the left leg.

After each of his attacks he slowly improved, and was usually soon able to walk with assistance or even alone, until August 29th, 1881, when he fell upon the floor of his room and broke the neck of the left femur, after which, of course, he never walked again, but sat in a chair or lay in bed. Within two years and a half after the original onset of his illness, he had twitching of the right leg and trembling, which was often so violent as to shake the bed, and stiffening of the right leg came on as he was getting about after the attack of paralysis in May, 1880. May 3d, 1885, he was slightly out of his head, and had a constant tendency to fall over to the left side in his chair. June 7th, 1885, it was discovered that he had gangrene of the left heel, which soon extended to the toe, and then increased in extent until it involved about half of the foot. In August, a line of demarcation formed, and the gangrene never extended after that time.<sup>1</sup> There was general cutaneous hyperæsthesia for about two months before his death, which took place October 10th, 1885.

Post-mortem examination was made about thirty hours after death. The spinal cord was removed. Upon opening its membranes, nothing unusual was observed until the upper lumbar region was reached, when a mass was found with the appearance, upon section, of a cyst containing blood; this was attached to the dura

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<sup>1</sup> The gangrene was of the dry variety, and there never was the slightest odor connected with it.

mater upon the left side. The cord, upon section, at its upper portion seemed softer than usual, and the cut surface presented a pitted appearance. The degree of softening increased progressively from above downwards until the lower dorsal region was reached, at which position, upon section, it was seen to be almost semifluid. The lumbar cord in its upper portion was much broken down; below again it seemed much firmer. The softening was below the mass mentioned, and attained its maximum degree about three-quarters of an inch below the mass.

On removing the skull cap, the dura mater was found to be tightly adherent to it throughout, so that it was removed with difficulty. On examining the dura mater, some spots seemed thicker than others, and there were two dark-reddish œdematous-looking spots upon the left side. Upon the surface of the brain corresponding to the described areas there were serous effusions in the arachnoid. The general surface of the brain presented no unusual appearance except that the convolutions looked flattened and narrowed; its surface seemed rather firmer than usual. The pia mater could be stripped from the brain at any portion with perfect ease, coming out without difficulty even from the deeper sulci, and showing the size and outlines of the convolutions. The inferior surface of the brain, when examined from before backwards, presented nothing of note until the crura cerebri were reached, when it was seen that the left crus was universally darker in color than the right, its inner edge being for about one-fourth of an inch dark slate-colored and ill-defined. Upon section, above the corpus callosum on the left side the white matter appeared to be normal. When the left lateral ventricle was opened, there were seen upon the surface of the corpus striatum two areas of softened matter which were dark gray in color and almost translucent and very soft. The optic thalamus also showed two spots of softening. Upon section of the corpus striatum, its middle portion was found to be converted into a cavity containing serous fluid, although the anterior

and posterior portions looked more natural. The inner portion of the optic thalamus was soft and reddish. The right corpus striatum and optic thalamus seemed to be natural.

When the thorax and abdomen were opened, it was seen that the amount of adipose tissue was unusually great, both in the walls of the cavities and in the omentum, and the cavities seemed to be too large for the organs, which were all shrunken and small. The lungs crepitated throughout, and there were no adhesions upon the right side. The left lung was somewhat adherent at its posterior middle surface. The heart was small, and its tissue very dark colored. The aortic opening contained calcareous deposits which were most marked upon the intercoronary flap of the valve. The aorta was about twice the natural size, being atheromatous and stiff, but not pouchy. The mitral curtains were somewhat thickened, but otherwise healthy. The liver was very small, and of a dark nutmeg color; its surface rough, and the capsule thickened. Upon section, the tissue of the liver for a quarter of an inch beneath the capsule was seen to be lighter colored than the deeper portion, which was very dark. The gall-bladder was adherent and thickened, and contained a gall-stone three-quarters of an inch in diameter. The kidneys were small and had a markedly urinous odor, and were dark nutmeg-colored. The capsules were thickened and removed with difficulty, and the veins upon the surface were well marked.

The left femur was found to have been fractured at the neck, partly within and partly without the joint, and there was much shortening of the bone, and the seat of fracture was surrounded with a large amount of callus.

#### REPORT OF MICROSCOPICAL EXAMINATION.

*Heart.*—The muscular fibres were apparently smaller and narrower than normal, although this was not determined by any comparative measurements. Quite numerous young connective-tissue cells appeared between the fibres. Small

deposits of yellowish-brown pigment were present in the muscle fibres, chiefly situated at either pole of their nuclei.

*Kidney.*—There was an increased amount of intertubular connective tissue, and often rich collections of dark-stained nuclei. The arterioles had thickened walls, especially the muscular coats, while the increased fibrous coats gradually united with connective tissue around, which was often well supplied with nuclei. The cells of the uriniferous tubules were granular and indistinct. The process was the early stage of interstitial nephritis.

*Liver.*—The lesions of a well-marked fatty infiltration were present. Slight increase of fibrous tissue, and in many acini indistinct and really granular cells were also present.

*Nervous System.*—Parts taken for examination: third left frontal convolution, left optic thalamus and corpus striatum, left crus cerebri, right optic thalamus, pons, medulla, spinal cord, and small tumor attached to meninges opposite the upper lumbar cord.

The specimens were hardened for section by placing them for eight weeks in a mixture of three parts of Müller's fluid to one of methylic spirit, with frequent changing then in dilute alcohol for one week, and finally for forty-eight hours in absolute alcohol.

These portions were permeated with paraffin, sections cut and pasted on the slides, the paraffin dissolved, and the sections stained in situ, thus reducing any distortion of the relations of the parts to a minimum. The stains were carmine, picro-carmine, and the double stains carmine and sulpho-indigotate of potash. The finer sections from which this study has been made were cut by Dr. Wm. M. Gray, microscopist of the Army Medical Museum.

*Convolution.*—There was a general enlargement of the capillaries, which were distended with blood, and surrounded by dilated lymph spaces. The larger vessels were similarly engorged with blood, and in places rupture of the wall had taken place and allowed an escape of blood-corpuscles into the surrounding tissue. The arterioles presented dis-



tinctly thickened walls—the thickening was of the coats outside the intima, and each vessel was surrounded by an open space caused by absorption and atrophy of the surrounding tissue. The edges of these spaces were frayed and usually more deeply tinted than the adjoining structures, and in the immediate neighborhood round and oval bodies, probably amyloid, were often noticeable. The ganglion cells were cloudy, often showed beginning pigmentation and poor staining, and in places were positively shrunken.

*Left Optic Thalamus and Corpus Striatum.*—There was an extensive degeneration of the normal elements. Such ganglion cells as remained were shrunken and cloudy, while in the major portion of the diseased area there was an entire absence of nervous elements, which had been displaced by numerous bands and bundles of sclerotic tissue that crossed and recrossed one another, leaving intervening spaces until the whole resembled a coarse lace-work texture. The blood-vessels were distended with blood, and showed thickened walls. In the less diseased parts there were collections of small round cells and a very general increase in the neuroglia.

*Left Crus Cerebri.*—The cells of the substantia nigrica showed the usual dense pigmentation, perhaps more so than normal. Their processes were shortened or absent. Anterior and posterior to this region distended vessels—sometimes with thickened walls, and here and there in relation to extravasations of corpuscles—were noted.

*Right Corpus Striatum, Medulla, and Pons.*—Appearances similar to what have been described were present in less marked degree.

*Spinal Cord (cervical and dorsal).*—The central canal did not exist as a channel, but was plugged up by its proliferated epithelial elements. In its immediate neighborhood, in the gray matter, extensive thickening of the walls of the smaller arterioles and capillaries existed, producing the appearances of *hyaline degeneration*. This pathological change was present in all degrees from the slightest thickening up to aggregated masses which surrounded the

vessel tube. The ganglion cells were ill-defined, shrunken, unequally stained, and often markedly pigmented. Their processes were almost universally shortened and frequently absent. In addition to spaces around the enlarged vessels, radiating fissures occurred, especially in the posterior gray horns, having no relation to vessel walls. No positive change in the nerve tubules of the white matter was present.

The membranes of the cord showed in places collections of inflammatory cells and vessels with thickened walls.

*Tumor.*—This was composed of an external envelope of well-felted, wavy connective tissue, through which a few young cells were scattered. Internally were the remains of a tissue composed partly of small round cells, like granulation tissue, and partly of blood-corpuscles and yellow pigment granules. The growth was probably a gumma.

*Cord Opposite the Tumor.*—The cord here showed extensive destructive changes. Nerve-cells and tubes were destroyed, wasted, and pressed apart. Numerous round and oval bodies were scattered through the degenerated tissue, which may have been amyloid, or, as Kesteven has suggested, ends of broken nerve-fibres. The hyaline change in the vessel walls was here seen in its highest perfection, so much so as, in some instances, to have entirely occluded the vessel lumen.

*Résumé and Remarks.*—From this study it is evident that the lesions may be arranged as follows :

1. Coarse, destructive disease of all tissues of the left corpus striatum and optic thalamus, and of the lumbar cord where pressed upon by the tumor.

2. A very general lesion of the blood-vessels, which manifested itself in the brain by dilatation of the capillaries and thickening of the walls of the larger vessels, and in the spinal cord, especially low down, by a typical *hyaline* degeneration of the capillaries and smaller arterioles.

3. Scattered areas of degeneration, which were either in relation to enlarged blood-vessels, and contained blood-corpuscles which had escaped through rupture, or had no such relations, and have been described as radiating fis-

tures. The exact significance of these fissures is doubtful, and they may have been caused by the process of handling or hardening the specimen. Although Kesteven<sup>1</sup> has described "radiating fissures which result from loss of the connective which, in the normal condition, binds together the nerve fibres into bundles," it seems likely, in the present instance, they had no pathological significance. Not so with the areas relating to blood-vessels, which, at least in many spots, had undoubted pathological importance.

4. A quite universal change of the ganglion cells, which were cloudy, unequally stained, pigmented, and had their processes either shortened or lost. This appearance is not always as important as it at first thought seems, especially when it is remembered that the recent experiments of F. Kreyssig<sup>2</sup> show that poor staining and even vacuolation of cells may occur from putting tissues hardened in chromates directly into strong alcohol. This method had been avoided in the present case, and hence it seems fair to suppose the lesion had pathological significance.

*Remarks.*—The features of this case that seemed to make it worth reporting were that the man was under observation from the time of the onset of the first symptoms of secondary syphilis to his death, eight and a half years afterwards, and that there was an opportunity to make the most extended investigation of the post-mortem conditions. The history of the patient is sufficiently exact and minute with regard to the dates and mode of onset of the various attacks, but many of the finer methods of diagnosis were not made use of, as, for instance, any study of the electrical reactions and the more delicate methods of examining the conditions of the organs of special sense.

It is in a high degree interesting to compare the symptoms as they were observed during life in this case with the post-mortem appearances. There were three distinct attacks of hemiplegia, one accompanied with aphasia, and

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<sup>1</sup> "Observation of some points in the Path. Histol. of Spinal Cord." Trans. Internat. Med. Congress, 1881, Vol. I.

<sup>2</sup> Virch. Archiv, Bd. 12.

after each one great amelioration of the symptoms took place; then came on secondary muscular twitching and contracture, and finally gradual loss of intellectual power. It seems hardly likely that the cause of these successive attacks could have been the condition of the left corpus striatum, for the disorganization of that portion of the brain substance was so complete that most probably this was of comparatively recent origin, and the attacks of hemiplegia which occurred seven years before death were due to some other cause. The only other possible cause discovered was the minute extravasations of blood which, in certain areas, were found in the brain substance around the arterioles. The old disintegrated sac, which was found low down in the spinal canal, was probably all that remained of a gunma which had softened down under the administration of the large doses of iodide of potassium which this patient took for a long time. This tumor and the secondary softening of the cord which existed below it were the only coarse syphilitic lesions which were discovered. There must, of course, have been paraplegia as a consequence of this, but it was unobserved, owing to the multitude of other causes that existed to make the limbs motionless. It cannot be known at what period of the patient's illness this tumor came on, but this is a matter of no moment, for there was no true paraplegia either at the time when paralysis first came on or for a long time afterwards, as the patient continued to walk a little until he had the fall which has been mentioned, and which resulted in a fracture of the neck of the femur.

The fact is a most striking one, that, although at first sight Broca's convolution seemed to present no evidence of disease, outside of its partaking of the shrunken and hardened condition which was everywhere evident upon examination of the brain surface, yet the microscope revealed the existence of a condition of the arterial supply which, upon careful consideration, was exactly what might have been expected when the state of the patient's power of speech and mind were recalled. After the violence of the hemiplegic attack with aphasia, which occurred more

than six years before his death, had passed off, the power of speech seemed nearly normal, but from that time it gradually declined until, when he died, he had certainly not more than one-fourth the natural power of communicating his ideas by speech. His word memory was very poor, and he had great difficulty both in getting a mental grasp upon the word he was in need of and then in enunciating it, and his sentences were always short and ill-constructed, like those of a young child. At the same time that the aphasia gradually increased, it may almost be said that his intellectual powers "sclerosed"—he seemed to have less thought and less feeling, and became all the time less and less like a man, and more and more like an animal. Naturally a man of few words, he became positively morose in his silence, sitting for hours in his chair, glowering at nothing if undisturbed, and snarling a monosyllabic answer if asked a question. Still he had his pleasures, and one of his greatest was to listen to any conversation that might be going on within his hearing. Although nothing ever induced him to join in it, either voluntarily or if asked, still he would often show that his power of memory was fairly good by letting it be known that he recollected most of what had been said. This condition of almost complete aphasia, as it finally became, would seem to offer very strong evidence, if any further evidence is needed at this day, of the correctness of locating the speech centre in Broca's convolution, for the way in which the power of speech gradually declined through years, and the way in which the blood supply must have been gradually choked off by the slowly increasing vascular disease were most strikingly in accord.

No thoroughly satisfactory explanation of the cause of the successive attacks of paralysis, which are so common in cases of brain syphilis, seems as yet to have been suggested by pathologists, and yet it is one of the most marked clinical features of the disease. Syphilitic paralysis seldom comes on with the same suddenness as that caused by hemorrhage or the washing of an embolus into one of the cerebral arteries, nor is it usually so complete or



of such long duration. There is commonly some warning of the onset of an attack of syphilitic paralysis—for some hours or even for a day before, the patient feels strangely, or has headache, or there is partial paralysis of the part subsequently to become completely so, and this fact constitutes usually one of the most valuable and reliable signs to enable the physician to make a differential diagnosis between syphilitic and other forms of paralysis. It may possibly be that the explanation of the recurring attacks of hemiplegia in this case is to be found in the small extravasations which existed near the blood-vessels in some areas of the motor tract.

The general hyperæsthesia, which was a marked feature in the last few weeks, was fully accounted for by the meningitis which the post-mortem examination revealed. The existence of an unusually large bed-sore over the sacrum coincidently with the meningitis and disease of the cord, which has been described, would seem to go to show that it was due to trophic nerve influence, as it has been described by Charcot, although the likelihood of trophic influences being capable of producing such an effect has been doubted by Green.<sup>1</sup> It is a thing not very uncommon for old people, or those who have aged prematurely, to have general hyperæsthesia and bed-sores as the last chapter of some wasting condition which finally ends their lives, and this goes toward showing the hyperæsthesia and the meningitis, which certainly caused it in this case, to have had some more than casual relation with the occurrence of bed-sore. This mode of death of old people does not seem to be commonly known among clinical observers.

The striking and unusual features of the case may be briefly summed up as follows:

1. The fact that Broca's convolution, which, upon macroscopical examination, presented no evidence of disease, was found to have a degree of change in and around the blood-vessels which fully accounted for the aphasia.

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<sup>1</sup> "Pathology and Morbid Anatomy," by T. Henry Green, American edition, page 26.

2. The exact correspondence there was between the clinical history of the case, in regard to the constantly increasing aphasia and the slow decrease of the intellectual powers, and the condition of the blood-vessel system discovered upon microscopic examination—for the supply of blood must have been slowly cut off from year to year, thus gradually producing the results observed.

3. The possibility that the successive attacks of hemiplegia which occurred in the case, and which were always preceded by premonitory symptoms, as is usual in cases of brain syphilis, may have been due to the extravasations which were found around the blood-vessels at various areas in the motor tract.

## ON PARAPLEGIA FROM POTT'S DISEASE.<sup>1</sup>

By JULIUS ALTHAUS, M.D., M.R.C.P. LONDON,

CORRESPONDING FELLOW OF THE NEW YORK ACADEMY OF MEDICINE; SENIOR PHYSICIAN TO  
THE HOSPITAL FOR EPILEPSY AND PARALYSIS. REGENT'S PARK, LONDON.

IN this short paper I wish to draw attention to some points in the pathology and treatment of paraplegia induced by Pott's disease of the vertebræ which appear to me of physiological interest, and also of some practical importance; and in order to give, as it were, chapter and verse for what I am going to say, I will begin with a short account of a case of this disease which was some months ago under my care at the hospital, and which will serve to illustrate the principal points upon which I desire to dwell. This was the case of a florid-looking girl, aged 20, who had been in good health during childhood, and had not suffered from any manifestation of the scrofulous diathesis in the glands or other parts, nor had there been any phthisis in the family of the patient. There was no history of injury to the spine. She had been in domestic service until two years ago, when she had rheumatic fever, which confined her to bed for six weeks. Soon afterwards, however, she was well enough to take another situation, in which she remained for nine months. She then, apparently without any cause, began to feel pain in the right foot, which presently became very severe, and was followed by loss of power in the right leg. This disabled her from attending to her duties. Eventually the left leg was likewise affected by pain and paralysis, and the patient was then confined to bed, being

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<sup>1</sup> Read before the Neurological Section of the Academy of Medicine, Oct. 8th, 1886.

unable to sit, walk, or stand. She was admitted into a provincial hospital, where she remained under treatment for four months, but at the end of that time her condition had not improved, except that the pain was lessened.

On examination, I found that there was almost complete paralysis of the right lower extremity, from the hip downwards, the patient being only able to impress a very slight movement to the hip-joint. The muscles were flabby, but apparently not wasted. The faradic and galvanic responses of the nerves and muscles were obtained with great facility and an unusually low current strength; and there were no qualitative changes, the cathode closing contraction appearing before the anode opening contraction, and cathode closing tetanus being easily induced. The superficial reflexes were likewise unusually brisk, as pricking or tickling the sole or any other part of the limb excited considerable jactitations, not only of the right, but also of the left leg. The same was the case with the deep reflexes, for the limb was spasmodically thrown about, not only on tapping the ligamentum patellæ, but any point of the belly of the rectus femoris, the tibia, etc. Ankle clonus was easily excited, and continued for several minutes. There was, however, no tendency to spontaneous cramps or jactitations, and no muscular rigidity anywhere.

The foot was habitually cold and clammy, and occasionally quite wet; the coldness was rather less in the leg, and the thigh was tolerably warm. Round the ankle and on the instep there were large livid patches which disappeared on pressure. There was, however, not the peculiar appearance of glossy skin. The different forms of sensation in the entire limb were perfectly normal, there being neither analgesia nor anæsthesia to touch, pressure, or heat and cold.

The *left* lower extremity was in all respects less affected than the right. The patient could bend the knee and draw the leg a little way up, and had also some little power in the foot and toes; but all these movements were very feeble, and she was quite unable to stand on

the leg. The superficial and deep reflexes were exaggerated, but not to the same extent as in the right limb; and a higher degree of faradic and galvanic power was required for eliciting good responses of the nerves and muscles. The vaso-motor symptoms of coldness, etc., about the ankle and instep were likewise less developed; and sensibility in all its forms was perfectly normal.

Examination of the spine revealed an angular deformity of the ninth and tenth dorsal vertebræ, the spinous processes being prominent in the centre, while the transverse and oblique processes protruded laterally. The structures above the projecting vertebræ appeared to be thickened and indurated; but the swelling was not painful, nor tender on pressure or percussion. There had never been any pain in the spine. No fluctuation could be made out, and there was no sign of an abscess, either in the spinal region or in the psoas muscle, or elsewhere. There was no increase of temperature in the swelling; but the flexibility of the spine was much diminished. The commencement of the deformity had coincided with the appearance of the pain and paralysis in the right leg fifteen months ago.

There was no affection of the bladder and rectum, no tendency to bed-sores, and the catamenia appeared regularly every two months. All the other organs and functions of the body were normal.

Under these circumstances, the diagnosis could not be doubtful. We had evidently to do with caries, probably non-tubercular, of the bodies of two of the dorsal vertebræ, with their intervertebral cartilages and accessory ligaments; while the contact of the diseased structures with the dura mater had led to an inflammation in the external layers of that membrane, which had resulted in thickening and the formation of cheesy products pressing on the spinal cord. In the latter, there was probably some degree of interstitial myelitis set up, more especially in the crossed pyramidal strands, as shown by paralysis and increase of deep reflexes, and also in the posterior columns, as shown by the severe neuralgic pain in the feet, of which

the patient had complained more especially in the beginning of the affection; while the central gray matter of the cord had probably remained normal.

The patient was at first treated with large doses of iodide of potassium and cod-liver oil, and friction of the paralyzed limbs with ammonia liniment. The result of this treatment, however, which was carried on for nearly three months, was absolutely *nil* so far as progress towards recovery was concerned, although it may have checked the further progress of the disease. I then requested Mr. Pearce Gould to apply the actual cautery to the spine on both sides of the deformity; and this was very thoroughly and effectually done by him on four separate occasions. After the second application there was a decided improvement in the paralysis, and after the fourth the patient had so far recovered that she was able to walk about as usual. The vaso-motor symptoms of coldness and lividity, and the excessive perspiration in the feet, had then disappeared, and the superficial and deep reflexes were almost normal, although still somewhat exaggerated. The patient was kept under observation for another six weeks, and as she continued well, was discharged from the hospital and sent to a convalescent home.

Patients suffering from paraplegia owing to Pott's disease have sometimes recovered by the opening of abscesses either near the seat of the disease or in the psoas muscle, or elsewhere. In the present case, however, there had never been any sign of an abscess; and, as the patient had been treated for a considerable time, both in the country and in London, by rest in the recumbent position and other measures without any improvement in the paralysis, we cannot doubt that it was the powerful counter-irritation set up by the use of the actual cautery which was really the curative agent in the present instance; for soon after it had been applied improvement was noticed, and this went on steadily to a cure under repeated applications of the same agent. We must therefore assume that under its influence the inflammation in the bones and the dura mater was

arrested, that there was absorption of morbid products which had been deposited in the external layers of the membrane, whereby the compression of the cord was relieved, and the interstitial myelitis which had been set up in several of the white columns of that organ was likewise cured.

The first symptom in the present case was pain in the right foot, which came on suddenly and was exceedingly severe; on the other hand, there had never been any pain in the back. The pain in the foot, which, according to the description given of it by the patient, appears to have resembled the lightning pains of locomotor ataxy, was evidently caused by the inflammation which then commenced in the dura mater and the cord, as it almost coincided with the beginning of the paralysis. Was the pain in the foot owing to irritation of the nerve-roots in their exit through the inflamed membrane, or to irritation of the membrane itself, or to myelitis? As the inflammation of the dura mater in the present case was most probably confined to its anterior aspect, where the anterior or motor roots emerge through the membrane, it is not likely that it was owing to irritation of these roots, which would have produced spasm rather than pain. Was it then owing to inflammation of the dura? Here it is worthy of remark that, although Kölliker has found no nerves in that membrane, Ruedinger has been able to trace them; and Vulpian has experimentally shown that the dura, although only slightly sensitive in health, may become highly sensitive when irritated or inflamed. Indeed the membrane only needs exposure to the atmosphere for a few hours to become exceedingly irritable; and when an irritant substance is thus applied to it, the animal yells with pain, while the pupils are dilated each time that the membrane is touched. The pupil being probably the most delicate æsthesiometer, any dilatation of it under the influence of an irritation of a tissue, shows that the latter is sensitive. I do not think, however, that the pain in the present case was owing to inflammation of the dura, which would no doubt have caused local pain and tenderness; and I am therefore in-

clined to attribute the violent excentric pain of which the patient has complained to irritation of the posterior columns of the cord.

The symptoms of coldness, lividity, and excessive perspiration about the ankles and insteps showed that there was some degree of vaso-motor as well as secretory paralysis combined with spasm of the arterioles. Vaso-constrictor as well as vaso-dilator fibres are known to pass through each segment of the spinal cord and run, most probably, in the lateral columns of the organ. The co-existence of paralysis in the present case, owing, as I shall presently show, to disease of a portion of the lateral columns, thus renders the interference with vaso-motor function which was observed easily intelligible. Patches of venous congestion have the same significance as excessive perspiration; for Claude Bernard has shown that irritation of the fibres supplying the sweat-glands causes arrest of secretion, while paralysis of these fibres leads to excessive sweating. Systematic rubbing of the legs and feet with stimulating liniments did nothing whatever to modify this condition, thus plainly showing that it was caused by central disease.

It was formerly believed that the paralysis which is so frequently seen in Pott's disease was owing to softening and actual destruction of the spinal cord. This view, however, seemed to be inconsistent with the recovery of power, which sometimes occurs even without active treatment. In order to reconcile this apparent discrepancy, Mr. Shaw has suggested that, after the portion of the cord corresponding to the diseased vertebræ has been destroyed, the continued bending down of the column into an angle allowed the sound parts above and below to come into contact and displace the diseased part, so as to unite and be able to resume their function. He also believed another ground for entertaining hopes of recovery to be, that the loss of substance in the cord was usually confined to the anterior columns; and as these constituted only a small portion of the cord, they would be more easily replaced by healthy structures. Both these theories appear to me extremely hazardous, and not in consonance



with the teaching of modern physiology and morbid anatomy. A portion of the cord which has once been actually destroyed can never again act as a centre or path for voluntary power, nor can we suppose that such a loss could be replaced, even at best, by anything else but connective tissue. Moreover, it is not at all the fact that only the anterior columns of the cord suffer from the compression. Indeed, if it were so, it is unlikely that paraplegia would result; for the crossed pyramidal strands, which we know to constitute the motor paths in the cord, do not course in the anterior portion of that organ, but in the posterior part of the lateral column close to the posterior gray cornua.

Another important and suggestive fact is, that a similar form of paralysis as in Pott's disease is caused by pressure from hydatid cysts, aneurism, abscess, or malignant disease, invading the organ from behind or laterally. It is, therefore, far more rational to assume that the entire cord suffers some degree of compression in paraplegia from Pott's disease.

That simple pressure on the cord may cause paraplegia has been experimentally shown by Vulpian, who introduced a small bit of a wooden match under the arches of the lower dorsal vertebræ in a guinea pig, and thereby caused paralysis in the hind legs, but without loss of sensibility or reflex motility. A quarter of an hour afterwards the bit of wood was withdrawn, and in another hour it was found that the hind limbs had recovered their motive power. It is, however, not in this simple way that paraplegia is produced in Pott's disease. Echeverria, Michaud, Charcot, and others have, on the contrary, shown that there is habitually inflammation of that portion of the dura mater, more particularly in its external layers, which corresponds to diseased vertebræ, and that this inflammation may spread both upwards and downwards beyond the seat of the caries. It is frequently confined to the anterior portion of the dura, but may extend through its whole circumference. The arachnoid and pia mater are frequently healthy, but interstitial myelitis seems always

to be present, and may affect, not only the white columns, but also the gray matter; and it is in severe cases followed by secondary degeneration of the crossed pyramidal strands below the seat of the lesion, and by ascending degeneration of the postero-internal or Goll's columns, which latter is chiefly seen in the cervical portion of the cord. There could be no doubt that in the present case the crossed pyramidal strands were affected, either by interstitial myelitis or by a degree of secondary degeneration—more probably the former, as shown by the symptoms of paralysis combined with exaggeration of the deep reflexes; and this myelitis was evidently more severe in the right than in the left half of the cord, as the two symptoms just mentioned were much more marked in the right than in the left lower extremity.

How have we to explain the fact that, while there was almost complete motor paralysis, at least in the right limb, yet there was no loss of sensibility? This is a difficult question which has not as yet received a satisfactory answer, and which I will now proceed to consider.

Taking the clinical symptoms of Pott's disease in its different aspects as a guide, it appears to me that we may distinguish *three different degrees of pressure* and subsequent morbid changes as far as the cord and the spinal membranes are concerned. We meet with cases of Pott's disease in practice where there is considerable deformity, but no paralysis, or any other nervous symptoms referable to the spine. In such cases I believe that the pressure which no doubt exists is only just sufficient to displace the spinal liquid in which the cord floats, but leaves the membranes and the substance of the cord itself intact.

*The second degree* of pressure I would assume to be that which exists in the case which I have just related, and where we have to do, not only with displacement of spinal liquid, but also with a moderate amount of external pachymeningitis and interstitial myelitis, causing paraplegia, but no loss of sensibility or reflex motility, and affecting therefore chiefly the white columns, but not the central gray matter. The posterior columns are not by any

means the only channels by which sentient impressions are conveyed, and sensibility will persist as long as a small portion of the nerve cells in the gray centre of the cord remains active. This fact was demonstrated by Schiff and Brown-Séquard. It has been suggested by Vulpian that white nerve-tubes are more easily compressed than gray cells; but, although this appears possible, it cannot be said to have been proved. There can, however, be no question that motion suffers more readily from pressure and other morbid influences than sensation; and that, where both functions have suffered in consequence of disease of the nervous centres or peripheral nerves, sensation is more apt to return, and more liable to be re-established by therapeutical procedures, than motion. In that form of paralysis of the musculo-spiral nerve which is clinically seen after contusion of that nerve, there is rarely anæsthesia, although the paralysis may be almost or quite complete. Hemi-anæsthesia from cerebral lesion is more easily recovered from than hemiplegia, owing to a similar lesion. In the last stage of tabes dorsalis, when the patient may be completely paralyzed and anæsthetic in his lower extremities, powerful faradization of the skin with a wire brush will often, at least temporarily, restore sensation. All these facts tend to show that, other things being equal, motion is more easily lost than sensation, and that the latter, even when its ordinary channels are closed, may be more readily transmitted along other channels than motion which sticks to certain hard and fast lines. This is also proved by the fact that, as far as I know, there is not a single case of Pott's disease on record where there has been loss of sensation while the power of motion remained; while the reverse is a very ordinary occurrence.

*The third degree* of pressure and its consequences in Pott's disease I assume to be that where we have to do with complete transverse myelitis, affecting not only the white columns, but also the entire gray centre of the cord at the level of the disease of the vertebræ, and being then accompanied with secondary degeneration of the pyramidal strands below the lesion and ascending secondary

degeneration in Goll's column in the cervical region. The necessary consequence of this must be not only paraplegia, but also anæsthesia and analgesia in all parts below the lesion, with paralysis of the bladder and rectum, and tendency to bed-sores. A case of this kind has been recorded by Buzzard in the "Transactions of the Clinical Society" for 1880. His patient died of obstruction of the bowels, apparently brought on by the application of a Sayre's jacket, and after death the mid-dorsal region of the cord was found to be completely disorganized. It is, therefore, seen that, with regard to prognosis, *loss of sensibility in a case of Pott's disease is a far graver symptom than paralysis*, inasmuch as it shows a more severe degree and a greater extent of myelitis, which then appears to have affected the entire transverse section of the cord. We know that this condition leads not only to complete paralysis and anæsthesia in all parts below the lesion, but also to paralysis of the bladder and bowels, deep bed-sores, and blood poisoning, thus placing the life of the patient in the greatest jeopardy. It is obvious that recovery must be impossible when such severe lesions have been produced; and I would therefore recommend an early resort to the actual cautery in all cases of Pott's disease where symptoms of paraplegia are beginning to be developed.

In conclusion, I will say a few words about the use of the cautery in spinal affections, and the vicissitudes which it has undergone in the course of time in medical estimation. During the first half of the present century, this agent was quite indiscriminately used in all spinal affections of whatever nature, and patients often underwent a great amount of torture without deriving any corresponding degree of benefit from the same. Romberg was the first to object to this treatment in cases of tabes in which he had found it quite ineffective, and probably in consequence of his protest, the use of the cautery declined a good deal during the next twenty or thirty years. My experience is, that the cautery does no good in affections of the substance of the cord, such as tabes, spastic spinal paralysis, multiple sclerosis, infantile paralysis, progressive

muscular atrophy, etc. ; but that it is of very considerable service in disease of the spinal membranes from whatever cause. The paraplegia of Pott's disease has recently been more especially treated with Sayre's jacket and large doses of iodide of potassium, which I believe to be inferior in efficacy to the cautery ; and I shall, therefore, be much gratified if the use of the latter will now be again more generally resorted to than it has been of late years in cases of paralysis from Pott's disease.

## CLINICAL NOTES ON CHRONIC LEAD POISONING.

By J. J. PUTNAM, M.D.

BOSTON.

THREE years ago, I reported to the American Neurological Association a series of eight cases of chronic lead poisoning, not presenting the signs usually considered characteristic of that disease, and therefore likely to escape detection if the indications set down in the text-books were strictly followed.

Since that time, I have continued these researches with the valuable aid of Prof. E. S. Wood, of the Medical College, and of Mr. A. M. Comey,<sup>1</sup> Assistant in the Chemical Laboratory of Harvard University at Cambridge, and have become more convinced than ever that chronic lead poisoning, especially from drinking water, is far more common than is usually believed.

It was my intention to select the cases for this new series almost at random, though giving preference, for therapeutic reasons, to those which exhibited the symptoms already shown to be significant. I hoped in this way both to test the frequency of occurrence of lead in the urine in cases not presenting the usual lead symptoms, or even, necessarily, any symptoms, and also to see whether any new symptoms or types of disease could be found to be characteristic.

I am well aware that cases are to be found scattered through the vast and unwieldy literature of lead poisoning,

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<sup>1</sup> Most of the analyses for this series were done by Mr. Comey, to whose skill and patience I am greatly indebted.

similar to most of those recorded in this paper, but I report them, first, because I believe it is not sufficiently recognized that no obscure case of functional or organic nervous disease should be thought fully investigated until lead has been sought for, and further, because I think it can be shown that lead is found with such relative frequency in certain forms of chronic myelitis that it may fairly be regarded as an important cause of that disease, all the more important because of the possibility of avoiding or removing it. Although a number of isolated cases of this kind are on record, yet no author of a text-book, so far as I am aware, speaks of lead as a frequent cause of chronic myelitis, except Dr. S. G. Webber,<sup>1</sup> in his recent manual.

It is especially desirable to know what symptoms should arouse even a suspicion of lead poisoning, because the analysis of the urine is so difficult that it can never be undertaken as a simple matter of routine, and we are generally forced to rely on the clinical evidence alone.

Finally, the investigation seems to make it clear that lead may be occasionally present in the tissues in sufficient quantity to make its detection in the urine possible, and yet exert no markedly injurious effect upon the health.

Thus, in a case of neuritis, involving both the lumbar and sacral plexus, and due to the pressure of cancerous growths, lead was twice found in relatively considerable quantity, although at the time the patient showed no sign of general cachexia or ill-health, nor any symptoms which our present knowledge would enable us to associate with lead poisoning.

The discovery of minute quantities of lead in the system, of course, does not justify us in laying to its score all the ills from which the patient may suffer, or in expecting necessarily to cure, by removing the poison, even the whole number of those symptoms for which the lead was directly or indirectly responsible. The best that we can hope for, at present, is to be able to say of lead, as we can

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<sup>1</sup> "Nervous Diseases," Boston, 1885.

now say of syphilis; first, that it sometimes sets up morbid changes which its elimination will cure; second, that by diminishing the resisting power of the tissues, it is the indirect cause of many trains of symptoms, a portion of which may disappear, or partly disappear, on its elimination.

The method of analysis of the urine, used in the former experiments and described in connection with the cases already reported, has been slightly modified for the present series.

In the first set, the lead was thrown down as an iodide, which was recognized by its yellow color.

This had the objection, the practical bearings of which were discussed in the paper alluded to, that bismuth, if present, would give almost the same reaction with lead. In the new series, therefore, Mr. Comey, at Prof. Wood's suggestion, has used the additional precaution of redissolving in hot water the iodides of lead and of potassium, after making the color test, and then filtering and precipitating the lead with dilute sulphuric acid, and allowing the precipitate, with the supernatant liquid, to stand for twenty-four hours in a test-tube wiped perfectly free from lint and other impurities. On then gently twirling the tube between the fingers, any sulphate of lead which is present rises in the form of a characteristic spiral cloud.

As regards the accuracy of the process, Mr. Comey says: "I tried three normal samples and found no trace of lead. I then added 0.005 lead acetate to the litre, that is one part in 200,000. In two instances, I found a slight trace of lead; in the third, it was doubtful if any was there. I then tried 0.020 to the litre, *i. e.*, one part in 50,000, and found lead far stronger than in any urine I have examined."

The symptoms of most frequent occurrence in the eight cases reported in the former paper were: muscular tremor; a sense of numbness and prickling in the legs or in all four extremities, with impairment of the strength; exaggeration of the knee-jerk, and other signs of diffused chronic myelitis. In one case, there was extreme and



progressive anæmia, with almost universal paræsthesia and gradually increasing paresis, especially of the legs, and eventually death. In another interesting case, in which the urine was twice found to contain lead, there were various diffused, bilateral cerebral symptoms, attacks of dizziness, headache, remittent "numbness," *involving all four extremities*; twitching of the muscles, or parts of muscles, on both sides of the body, but especially the left arm and leg, and steady improvement attended the use of small doses of iodide. The diagnosis was complicated by the fact that the patient had had a chancre, without secondary symptoms, ten or eleven years before, and I will here take occasion to remark that the early symptoms of cerebral syphilis and those of the cerebral cases of lead-poisoning are sometimes strikingly similar.

The whole number of new analyses of the urine on which I have now to report is forty-eight, and in twenty-five of them lead was found. Only those cases are included in which blue line on the gums, paralysis of the extensors of the fingers, and characteristic colic were absent, and, it might be added, with one or two exceptions, only those whose occupation would not have been thought to expose them to poisoning from lead.

Of the twenty-five cases in which lead was found, signs of diffused myelitis of one or another type were present in twelve. These twelve cases may be divided between those where the lead—if it was in fact the efficient cause—had excited typical groups of symptoms, mainly referable to localized lesions; and those where the symptoms were relatively diffused and irregular.

The former group embraces four cases, with symptoms pointing either to *multiple cerebro-spinal sclerosis*, or to *spastic paraplegia*, such as is usually considered due to primary sclerosis of the lateral motor tracts.

The spinal cases in which the urine was examined for lead and none was found<sup>1</sup> are eleven in number, and com-

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<sup>1</sup> It is worth remarking that a single negative examination of the urine does not prove that lead is not present in harmful quantities. In two of my cases,

prise four typical cases of progressive muscular atrophy, and two of locomotor ataxia—the only cases of these kinds that were examined at all; one case of subacute, symmetrical poliomyelitis of the adult; two cases of spastic paraplegia; one with symptoms of multiple sclerosis, but beginning in early youth; and one obscure and irregular case of diffused cerebro-spinal disease.

The four cases of sclerosis in which lead was found, and yet which presented few, if any, signs of the generalized action of the poison, were, in brief, as follows:

CASE I.—Male patient, married, 30 years of age, farmer. Presents symptoms of chronic malaria and has a large spleen; otherwise, general health excellent. During past two years, increasing weakness and awkwardness in use of all four limbs; feet and hands often numb; pains in various parts of body; loss of sexual desire, following increase; spastic gait, especially right, and exaggeration of deep reflexes; local anæsthesia in various parts of body, especially on right side; vision normal, fundus oculi normal; no history or sign of syphilis; *very small amount of lead in urine* (Prof. Wood). Occupation does not bring him into contact with lead. Drinks fresh pond water (Cambridge, Mass.), not drawn through lead-pipe. Until within a year, however, drank water from a deep well. Does not know what kind of pipe was used, but remembers that another person who drank from the same well had symptoms said to have been traced to lead. Marked improvement at first under treatment by iodide of potash, quinine, galvanism, and cauterization; *later*, relapse, cystitis, attacks of faintness, mental confusion, and drowsiness.

CASE II.—Male patient, bootmaker, married, 50 years of age. Spastic gait; exaggeration of the deep reflexes; ataxia of the hands; and permanent contraction of both pupils. *Lead was found twice in the urine*, at intervals of ten months, one examination being made by Prof. Wood, and one by Mr. Comey. No other cause whatever could be assigned for his disease.

CASE III.—Male patient, painter, married, 46 years of age. Spastic gait, with exaggeration of the deep reflexes; sudden attacks of dizziness; increased frequency of micturition; *small trace of lead in the urine*. No other cause could be assigned.

CASE IV.—Male patient, about 35 years of age. Symptoms limited to spastic gait, exaggeration of knee-jerk, and ankle clonus. No pain or disorders of sensibility; no paralysis or atrophy or disorders of micturition. Onset gradual, and dates back ten years;

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it was found only twice out of three trials. See in this connection a paper by C. B. Penrose, Phila. Med. News, Sept. 14th, 1885.

*considerable amount of lead in urine* (Prof. Wood). At time of onset was living on a farm and drank water drawn with wooden bucket; not long previous to this, however, he lived on a farm where he drank a great deal of water which came half a mile through a lead-pipe; gradual improvement, he thinks, during several months of treatment by iodide of potash, etc.; then lost sight of. No cause other than lead could be assigned for the disease, except a fall from his horse, some time before the symptoms set in.

In the absence of lead cachexia, marked debility, muscular tremor, or recovery under iodide of potash, there must remain a doubt, which can only be settled by many observations, whether the symptoms in such cases as these are really due to lead, and if so, what the nature of the relationship may be. It is, however, to be remembered that a similar uncertainty attaches to the nature of the connection between syphilis and locomotor ataxia; and yet, nevertheless, the fact that there is some connection is almost beyond a question.

In the remaining cases, although the spinal symptoms predominate, a wider and more diffuse action of the poison is indicated, and in most of them more or less signs of disturbance of the general nutrition were present. The first case approaches closely those of the former group.

CASE V.—Male patient, sawyer, married, 49 years of age; lives at Somerville, Mass. Sense of weakness and pain in back for ten or twelve years, but worse during the past two years, without apparent cause; sense of fatigue in legs and thighs on slight exertion; spastic gait, general feeling of drowsiness and languor; cramps in calves of legs; feeling of soreness along both groins and in the back; arms and hands rather weak; no alteration of sensibility, subjective or objective; face pale; pupils large and mobile; heart-sounds weak, but no murmurs; sleeps fairly at night, but not so soundly as formerly; chancre twelve years ago, with doubtful secondary symptoms; exaggerated knee-jerk, ankle clonus; and some exaggeration of deep reflexes in upper extremities; strong trace of lead found in the urine; considerable quantity also in drinking-water.

CASE VI.—Male patient, married, 53 years old; lives in Salem St., Boston.

Sense of numbness and prickling in the hands and arms, alternating with sense of heat and cold; sometimes slight dragging of left leg and foot in walking. This feeling of numbness is most

marked in forefinger of left hand, which is also abnormally sensitive to heat, and is the seat of an unnatural sensation when any object is grasped; fingers twitch, and feel swelled toward night; two middle toes of left foot sometimes feel numb; is easily fatigued, and short of breath on exertion; all these symptoms worse towards night time; pulse full, 72, not tense; slight trembling of muscles of neck, and marked trembling of hands and arms; knee-jerk exaggerated both sides, especially left; is rather thin, but a sufficiently healthy looking man; no history or sign of syphilis; small amount of lead found in urine; has used hair dye for a number of years, but the coloring matter in this proves on examination to be made of nitrate of silver.

CASE VII.—Female patient, married, 45 years old, lives in Milford.

Debility; diplopia; dizziness; slightly staggering gait; headache, especially on right side; backache; right pupil slightly smaller than left, and reacts less to light; small atrophy of optic disc; exaggeration of knee-jerk and ankle clonus, especially right; lead found in urine.

These symptoms are attributed by patient to a severe injury received nine years ago. The case is reported because of the possible significance of the exaggerated knee-jerk as pointing to lead, the cerebral disease being hardly sufficient to explain them.

CASE VIII.—The following case is remarkably interesting, both on account of the character of the symptoms referable to disease of the spinal cord, and on account of the improvement under treatment. It will be published elsewhere at length, but is here given in brief outline.

Female patient, single, 27 years old. Health good until about three years ago, since when has suffered from progressive weakness in legs; later also in arms; aches and pains about the joints; marked impairment of the cutaneous sensibility, and marked sense of numbness; some muscular atrophy, especially noticeable in the ulnar distribution of both arms; blurring of vision with considerable impairment of vision, left; almost entire loss of muscular sense in the hands and arms, and a high degree of ataxia of movement; marked exaggeration of knee-jerk, and some increase of the deep reflexes in the upper extremities. It was found that the patient drank during part of the year from a very old well, the water of which was found to contain offensive organic matter, and a large quantity of lead. The urine also contained a considerable quantity of lead. The only other possible cause for the symptoms lay in the fact that one or two members of the family in a previous generation were said to have suffered in a somewhat similar manner. The patient was put on treatment of iodide of potassium, and, although she had been steadily losing ground previously, she gained steadily, and nine months later was reported

by her sister as being able to use her hands almost as well as ever.<sup>1</sup>

CASE IX.—Male patient, about 35 years old; not exposed by his occupation to lead. General feebleness for fifteen years; muscular weakness and sense of prickling and numbness in legs for the past few months, and some objective loss of sensibility; same symptoms in less degree in arms and hands; twitching and cramps in legs; history of malarial fever; lead found in considerable quantity in urine; marked improvement under treatment by potassic iodide.

CASE X.—Male patient, married, about 35 years old. Muscular tremor; exaggeration of knee-jerk; no ankle clonus; several attacks of numbness and prickling of tongue and mucous membrane of the mouth and hand on left side; later, slight attack of aphasia; no history or sign of syphilis; urine examined twice for lead; first time a considerable quantity found, second time none found.

CASE XI.—Male patient. Loss of power in the fingers and thumb of the right hand, at first remittent and associated with temporary attacks of numbness and prickling, then permanent. Later, temporary loss of vision in right eye, and two weeks later, a similar attack. Tenderness on pressure of toes of right foot; slight aphasia, and slight difficulty in articulation; no sign or history of syphilis; small amount of lead found in urine. *Works in a rubber factory and handles rubber in the making of which litharge is used.*

The following case was not included in the list, because the urine had not yet been examined. It is, however, recorded here, because the signs of lead poisoning are unequivocal. The interest of the case is the greater that this patient, like the last, worked in a rubber factory. This occupation has furnished to me, and to several of my colleagues, a number of cases of serious impairment of the health, though not all presenting the same symptoms. The room-mate of the patient whose case is about to be reported, suffers from symptoms closely similar to his, and was told by the physician whom he saw at the Massachusetts General Hospital that he was under the influence of some poison, my informant did not remember what. Litharge is largely employed in manufacturing articles of rubber, and although not used in the factory from

<sup>1</sup> Two cases of ataxia, more or less resembling that reported here, are quoted in the Monograph of Renaut ("De L'Intoxication Saturnine Chronique," Paris, 1875), from observations by Raymond and Vulpian.

which these last-mentioned patients came, their work consisted in grinding up articles made of rubber, and probably exposed them to dust containing particles of lead. I have heard from another source the general statement that the health of the operatives at this factory was not good.

CASE XII.—Male patient. Severe abdominal pains for the last four months; constipation and some colic; tenderness on pressure, in the abdomen; pain also in back; fatigue in walking; insomnia, due, he thinks, to pain; complexion and conjunctiva slightly yellowish; exaggeration of knee jerk, both sides, and traces of ankle clonus; marked blue line on gums.

This case, with its symptoms of typical lead-poisoning blue line, and colic or abdominal neuralgia, and at the same time with its signs of myelitis, the exaggerated knee-jerk, and trace of ankle clonus, seems to strengthen the belief that the cases where spastic gait and exaggerated knee-jerk formed the main symptoms, really owed their disease to the lead which was found in the urine. In fact, an abnormally active knee-jerk has come to be strongly associated in my mind, perhaps too strongly, with a suspicion of lead-poisoning.

In looking over the recent entries in the hospital records, I find one case with these notes: "spastic paraplegia; numbness of hands and feet; exaggerated knee-jerk; works in a rubber factory." The urine has not yet been examined for lead, but I shall seek an early opportunity to have this done, with some degree of confidence that the result will be affirmative.

CASE XIII.—Male patient, single, about 35 years old. Sense of fatigue in legs, felt mainly in the calves, so that he gets extremely weary at his work; no impairment of gross strength; sense of numbness and coldness in thighs, especially over anterior surface; calves of legs feel stiff and numb in the morning; has pain running down them as far as the heel, yet is not conscious of any stiffness of the legs in walking; remains of an acute cystitis for which he was treated recently at the City Hospital; tremor of hands and muscles of face in talking (this symptom, he says, he has had ever since boyhood, but for the past few months it has been better than ever before; a year ago it troubled him so much that he was sometimes thought to be drunk, and could scarcely sign his name distinctly); no sign or history of syphilis; physical

examination shows nothing abnormal except exaggerated knee-jerk; urine contains considerable lead. A possible source of the lead was a former habit of holding a piece of lead in his mouth.

CASE XIV.—Male patient; tremor of hands; constipation; “rheumatic” pains in arms and shoulders; drinking-water comes from a wooden tank, but urine contains a small amount of lead.

CASE XV.—Male patient; married; switch-tender, and much exposed to bad weather; habits of life unexceptionable in every respect. Has been under observation a number of years at the Massachusetts General Hospital, presenting the following symptoms: Constant aching-pains in back and legs, called “rheumatic;” stiffness and weakness of legs; fine tremor of hands; no disorders of micturition. The urine has been examined three times, with the result that lead was found in considerable quantity the first and last times, and was not found the second time. For a long period, under the use of potassic iodide, cauterization and galvanization of the back, there was marked improvement, but of late he has relapsed.

As I have already said, instances of paraplegia from lead are to be found here and there in the literature of lead-poisoning, though they appear to have attracted but little attention, as a rule. The report to the Am. Med. Assoc., in 1852, by a committee, of which Dr. Horatio Adams, of Waltham, was chairman, refers to several cases in which symptoms like those which I have repeatedly found had been described by their correspondents.

One case, which is narrated by the patient himself in full detail, is that of a prominent citizen of Boston, who suffered from symptoms closely resembling those of typical amyotrophic lateral sclerosis, with contractures to such a degree that he was almost confined to his chair, and yet greatly improved, temporarily at least, after discontinuing his drinking-water and taking heroic doses of strychnia. The urine, in this case, was not analyzed, but the patient says of his drinking-water “that that for cooking passed through 140 feet of lead-pipe to the kitchen pump, and that he had used it for nine years; that, for some months past, he had made his breakfast of crushed wheat, which, after soaking in water all night, was put on to boil the first thing in the morning, and probably the water which lay in the pipes all night was used for this purpose. The drinking-water chiefly used was brought through 140 feet of lead-pipe from another well to another pump.

“On being tested with sulphuretted hydrogen, that of the kitchen pump became very dark, and, after standing till the next day, looked like ink and water; that from the other pump was colored, but less highly.”

*Fine muscular tremor* was noted in several of the spinal cases, and in nine others, and was usually associated with debility or other characteristic symptoms. This muscular tremor is not to be confounded with the coarse trembling seen in later stages of lead paralysis; and, although by no means a new symptom, it is noteworthy as being of a great diagnostic significance even when unattended by any other signs of lead-poisoning whatever, and although continuing for years without being followed by paralysis or any other serious symptom, and even when so slight as scarcely to attract the patient's attention.

Such was the chief symptom, though, in each instance, associated with more or less general debility, in the following cases :

CASE XVI.—Female patient, somewhat past middle life. Debility; very fine tremor of hands; no other symptoms; lead found both in urine and in drinking-water.

CASE XVII.—Male patient, a business man in good circumstances. Debility; fine muscular tremor; small amount of lead found in urine.

CASE XVIII.—Male patient, 54 years old, weaver in a mill at Maynard, Mass.

Good habits in every respect, and excellent health until about three years ago, and even up to the present moment in most respects. Three years ago, he first noticed general weakness and a tremor of hands; for past eighteen months has suffered greatly from a feeling of soreness at the vertex and from attacks which he calls "rush of blood," and describes as a sort of aura spreading over the body and head; also from "buzzing" noises in the head. Sleeps well; no colic; no mental symptoms; no blue line.

The face is reddish and rather congested, and nothing in his appearance suggests the lead-cachexia, except the slight trembling of fingers, lips, and tongue.

On close and repeated inquiry, it was learned that the drinking-water was conducted to his kitchen pump from a well twenty-five feet away, through a two-inch lead pipe, and that the end of the pipe rested permanently in the well. Several other families were supplied from the same well.

The water was analyzed, and found to contain lead. The urine was analyzed three times, the result being twice affirmative of the presence of lead, and once, the last time, negative.

The patient had lived ten years in the same house. He had been in the habit of drinking a goblet of water every night, but



not, it is interesting to note, from the first pumping. The kettle for cooking had always been filled at night.

A sense of "*prickling and numbness*" in the extremities, usually intermittent or remittent, and worse at night, was observed a number of times, but I do not consider it so characteristic a symptom as the tremor, because it occurs under so many different conditions, of the nature of neurasthenia and general debility.

A similar condition, but associated with typical hemianæsthesia, and sometimes hemiparesis, has several times been reported as due to lead poisoning.<sup>1</sup> Recent researches, both at home and abroad, have, however, shown that this symptom, besides being met with in hysteria and as a result of lesions in the posterior limb of the internal capsule, is readily brought on by a variety of causes which tax the nervous system severely and suddenly, such as epileptic attacks, concussion accidents, and the like, and it is probable that its relation to lead-poisoning likewise is of this general character, and not the sign of a localized organic lesion.

It is quite possible that the same may be said of the *exaggerated knee-jerk*, which was so frequently observed in my cases. I have but little doubt that spastic paraplegia of organic origin, of which this is a characteristic sign, is often due to lead-poisoning; but in other cases, the lead, besides the organic lesions which it causes, seems to induce a sort of neurasthenic condition in which, as is well known, the exaggerated knee-jerk, without ankle clonus, is often met with. Such is at least a possible explanation of this symptom as it occurs in the following case:

CASE XIX.—Printer, 25 years of age; well until four years ago, since when he has suffered from increasing nervousness and sleeplessness; tremor of hands and lips; no excess of any kind, except perhaps tobacco; no sign or history of syphilis; no headache, colic, or constipation; no paralysis of sensation or motion; knee-jerk very lively, especially right; no ankle clonus; frequency of micturition; large amount of lead in urine; on a second examination, knee-jerk less marked.

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<sup>1</sup> Raymond, quoted by Renan: *De l'Intoxication Saturnine chronique*, 1875  
Hanot et Mathieu, *Arch. Gén. de Méd.*, 1873.

Another point is worthy of note, namely, that an inequality in the knee-jerk of the two sides also points to organic disease, either in the cord or brain or peripheral organs. Besides the cases which I have reported, I have notes of two cases of epilepsy,<sup>1</sup> beginning rather late in life, where lead was found; and a case of mixed intracranial and spinal disease, where the symptoms seemed to be mainly due to syphilis. One or two other observations also are not reported for want of sufficient notes.

CASE XX.—The last case that I shall refer to is that of a gentleman, naturally of robust health and free from neurotic tendencies, who has suffered for about a year from a severe form of clonic torticollis, associated with considerable depression of spirits, and for a number of years from muscular tremor of the hands.

Analysis of the urine and of his drinking-water showed the presence of lead in both. The drinking-water, which, I believe, was only used for a part of his supply, came from a cistern through a lead pipe, the end of which stood in the water.

The pipe was removed, to the great scorn of the plumber, and four inches of the end was sent to me for inspection. No corrosion could be detected, and the question arises whether, under these circumstances, sufficient lead could have been dissolved to exert a poisonous effect. The answer can probably be given in the affirmative. In the first place, lead is always dissolved by water containing air, acid, or organic matter in solution, unless it is protected by a coating of insoluble salts, mainly sulphates and carbonates. The very deposition of these salts, however, as is pointed out in a recent pamphlet by Hamon,<sup>2</sup> is probably an indication of the galvanic action along the walls of the pipe, since otherwise the salts would be deposited, by the action of gravitation, on the depending portions of the tube, instead of uniformly on its surface.

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<sup>1</sup> One of these two patients with epileptic attacks has just reported that on the second examination of urine, which has just been made, a considerable amount of lead was found. There is nothing in his appearance to suggest lead-poisoning except a look of slight debility; even tremor of the hands is absent. The importance of the discovery is obvious, the more so that isolated cases of the kind have been reported.

<sup>2</sup> "Étude sur les Eaux Potables et le Plomb." Paris, 1884.

It is probable that this galvanic action sets free lead in a soluble form. It has also been shown to be probable that the same galvanic action goes on, to greater or less degree, beneath the protective coating, so that sooner or later even cold-water pipes are sometimes eaten away.

The interesting paper on lead-poisoning in the Massachusetts State Board of Health report for 1871, by Prof. William R. Nichols, also contains a suggestion which is interesting in this connection. He says (page 33): "In all water, hard and soft, there appears to be formed at first an oxide (or hydrate), and this also is more soluble than the oxy-carbonate. If lead be partially submerged in water, there will always be found on it, after some days, *at the surface of the liquid*, yellowish-white crystals of hydrate of lead, along with the crystals of oxy-carbonate."

I have italicized the words "at the surface of the liquid" to call attention to the possible increase of danger where lead-pipe is partially submerged in a reservoir, the level of the contents of which is rising and falling. Whether such was the case here I cannot say. It is also well known that the galvanic action set up by lead-pipes which are soldered to stop-cocks or to iron mains is a fruitful cause of active corrosive action.

In all the cases that I have reported in this paper, the source of poison was unknown, except in those where it was specified. Did it come from the drinking-water, which in almost every cause was conveyed through lead-pipes? Certainly, the contrary cannot be asserted with confidence.

The last report of the Massachusetts State Board of Health, 1886, says that but few cases have been reported since the large number contained in the report for 1871. May this not mean that, owing to the increased precautions suggested by a better knowledge of the subject, the more extreme cases of lead-poisoning have been avoided, but not those characterized by less violent, but hardly less serious symptoms? In the report of 1871, Prof. Nichols says (page 37): "I feel justified in asserting that Cochituate water which has passed through lead-pipes is never ab-

solutely free from lead." In the pamphlet by Hamon, to which I have already referred, an account is given of some experiments made by Gautier and reported to the Academy of Medicine at Paris, in 1881, in which he found that the water of the Dhuis, after standing ten hours in a new lead-pipe, contained 0.0001 of lead to the litre. This is not far from one-hundredth of a grain to the gallon; and in the report made to the Am. Med. Assoc., in 1852, by Dr. Adams, this proportion of lead is declared to have several times proved injurious. A simple calculation shows, to be sure, that a person drinking daily one quart of water containing this proportion of lead would take only about one grain of lead in the course of a year—a quantity which seems absurdly small. But, while we are at liberty to doubt the accuracy of the chemical estimate, we are not at liberty to set aside the opinion that minute quantities of lead taken into the body little by little may, after a time, give rise to serious effects.

It is well known that, in all except the newest of our dwelling-houses, lead-pipe forms the connection between the street-mains and the houses, and it is difficult to find a satisfactory substitute. Galvanized iron, brass, and block tin, all have their objections, although the remark of a witty Frenchman may be put on record that "in the use of brass for domestic purposes the anxiety felt is out of proportion to the danger, while in the use of lead the danger is out of proportion to the anxiety."

The best substitute for lead is probably lead carefully lined with block tin, or rather superposed upon tin. Even this, however, is not absolutely free from danger, both because the coating may give here and there, and because of the danger of galvanic action in the joints. Furthermore, it would be out of the question to introduce this expensive substitute into the older portions of the city. In the face of these facts the question arises, what next can we do to secure a safe drinking supply? The answer would seem to be the one which has so often been given: first, that the water should be allowed to run from the pipes a sufficient length of time before it is used; and then

that cold-water pipes, used for drinking purposes, should not be allowed to run in the immediate neighborhood of the hot water service.

The next question is, how long must the water run in the morning in order to free the pipes of the contents which have accumulated during the night? A piece of lead pipe four inches long and an inch and one-fourth in diameter contains about two ounces of water, so that something over two gallons would be contained in fifty feet of pipe. It is probable that a good deal more water than this should be allowed to run off if we wish to be sure that that which remains is absolutely free from contamination.

In an interesting communication, by Dr. W. B. Thomas, in the *London Med. Press and Circular* of January 27th, 1886, a series of cases are reported of lead-poisoning from drinking water in the town of Sheffield, England. In relating them, the writer says that in each case the patients asserted that they had allowed the water to run five or ten minutes before using. It appeared evident to Dr. Thomas that the number of these cases had materially increased within the past few years, and, in his opinion, this increase was due to the fact that the water had recently contained more free acid than previously. In this connection, it is to be borne in mind that the solvent action of the water in our large cities, and still more in the country towns, is liable to be at any time similarly increased, either by the introduction of new sources of supply, or by the temporary increase in the organic impurities washed in by large rain-falls, or due to the contamination from factory towns.

The greater part of the cases reported in this paper are those of patients living in the country. A few, however, were from Boston and the neighboring towns.

I intend, as soon as possible, to carry these investigations further, especially with regard to the cases of this latter group. May we not also hope that something still further can be done for the protection of workmen engaged in occupations where the use of lead exposes them to life-long disablement, or that, at least, some way should

be found to give them a more intelligible warning than they now receive of the danger to which they will be liable.<sup>1</sup>

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<sup>1</sup> I desire to express my obligation to my colleague at the Mass. Gen. Hosp. Dr. G. L. Walton, for allowing me the use of his notes of several of the cases reported. I wish to say in addition, that although I have given the cases in brief outline only, the patients had been examined carefully for other causes of disease, especially chronic nephritis and syphilis.

## Clinical Cases.

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### A CASE OF PROFESSIONAL NEUROSIS OF CO-ORDINATION OF UNUSUAL ORIGIN.

By PHILIP COOMBS KNAPP, A.M., M.D.,

PHYSICIAN TO OUT-PATIENTS WITH DISEASES OF THE NERVOUS SYSTEM, BOSTON CITY HOSPITAL.

The symptoms which are classed under the various headings of professional neuroses of co-ordination (*co-ordinatorische Beschäftigungsneurosen*, Benedikt), professional hyperkineses (Ross), ana-peiratic paralyses (Hammond), *copodyscinesia* (Lewis), the neural disorders of writers and artisans (Lewis), or, in common parlance, the various "cramps"—writers', telegraphers', or pianists'—may arise, as is well known, from almost any occupation requiring protracted use of the muscles. The commonest form, which is regarded as the type of the affection, is writers' cramp, and next in frequency comes telegraphers' cramp, which attacks especially those who use the Morse key. Beside these comparatively common forms, there are numerous others, which are quite rare and are of interest mainly as curiosities. Among these are hammer-man's palsy, pianists', milkers', violinists', cello-players', and ballet-dancers' cramps. Dieulafoy cites a case of cramp in a cellar-man, which was due to his habit of giving a slight rotation to a champagne bottle whenever he served champagne; Poore<sup>1</sup> tells of a watchmaker who could not hold the lens in his eye, and of a pickle-jar tyer who could not hold the cover of a jar; and Lewis tells of a woman who had cramp in the fingers, which, as she was blind, she used for reading the raised type. Although the hands and arms are the parts most often affected, the cases cited above show that other parts may also be involved. Zenner's recent case<sup>2</sup> of auctioneers' cramp shows, also, that the lips and tongue may be affected, and spasm of the larynx has been observed in clarinet-players (Eichhorst).

As Lewis says,<sup>3</sup> "all occupations which require a muscle or a

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<sup>1</sup> Lancet, August 21st, 1886.

<sup>2</sup> Reported to the American Neurological Society, 1886.

<sup>3</sup> Pepper's "System of Medicine," vol. v., p. 511.

group of muscles to be kept in a constant more or less firm contraction, together with fine movements of co-ordination in themselves and in the neighboring muscles, may be expected to furnish cases of this class of disease; the muscles affected necessarily varying with the work done, mere routine work being more liable to cause trouble than that which is new and original, as in the latter case time has to be taken to elaborate it, thus giving temporary rest to the muscles." It would seem that the tonic contraction of the muscles is more injurious than the rapid and fine movements of co-ordination; for Poore, who has made a careful study of the muscles affected in writers' cramp, claims that the muscles of prehension are first affected.

The pathology of the disease is still obscure. Erb,<sup>1</sup> some years ago, said: "In the present state of our knowledge, we are justified in placing the seat of the cause of the typical forms of writers' spasm in the central nervous system, although we are not in a position to locate it with precision. Whether the trophic disturbance is to be sought for in the gray substance of the cervical portion of the spinal cord, or in the cerebral peduncles, or, lastly, in the gray substance of the brain, can only be determined by future investigation." Many writers claim that the disease is of central origin, but others think that it is at first peripheral, but by abuse it may become central (spinal). "The latter theory," says Lewis again,<sup>2</sup> "and not the idea that it is a disease of the co-ordinating centres in the brain, or of the spinal centres only, best explains, in my opinion, the various symptoms encountered. . . . Exactly what the alteration in the condition of the spinal cord is which probably occurs in many of these cases it is impossible to state, but the view that it is a nutritive change of the upper dorsal and lower cervical portion of the spinal cord (that is, when the arm is the part affected, as it is in all but the rarer cases) is quite attractive, the condition being secondary to a peripheral irritation in many cases." Weir Mitchell states that subacute neuritis is often incapable of distinct clinical discrimination, when of a mild type, and when there is an absence of traumatic cause. Eichhorst says<sup>3</sup> that "the disease is probably the result of purely functional disturbances. These are evidently situated in the spinal cord, because the act of writing requires the delicate co-ordination of adjacent groups of muscles, and the co-ordinating centres of these muscles are situated in the cervical enlargement of the cord. Abnormal irritability, ready exhaustion, and irregular stimulation of these centres suffice to explain the symptoms of writers' cramp. In some cases, however, the primary disturbance is situated in the brain, as is shown by the feeling of pressure in the head, vertigo, mental depression, etc. In

<sup>1</sup> Ziemssen's "Cyclopædia," vol. ix., p. 155.

<sup>2</sup> *Op. cit.* pp. 528, 530.

<sup>3</sup> "Handbuch der speciellen Pathologie and Therapie," Bd. ii., p. 566. Ed. 1883.



other cases, the starting-point is at the periphery (neuritis, periostitis, etc.).” The case which I shall report seems to be of this latter type, perhaps one of the cases of neuritis of which Weir Mitchell speaks.

The symptoms of these professional neuroses are not by any means all of the same type. Benedikt has arranged them under three classes : the spastic, the tremulous, and the paralytic forms. Of the latter, Erb says :<sup>1</sup> “ Fatigue and weakness of the hand constitute prominent symptoms, in which there are no distinct spasms, but in which paralysis is more or less well marked, though, perhaps, it is only observable when the patient attempts to write. A gradually increasing and very decided sense of fatigue is experienced in the hand and forearm, which become, as it were, stiff and are no longer capable of being moved ; pain is felt in the whole arm, and, if the act of writing is persisted in, it extends to the shoulder and back.” This form is said by Eichhorst to be the rarest.

Although this classification is the one generally accepted, Lewis has proposed a more elaborate one, under one form of which the case I am about to report can better be placed than under Benedikt's paralytic form. He classifies the symptoms under five heads : cramp, paresis, tremor, pain or perversion of sensation, and vaso-motor and trophic disturbances. Under the fourth head, he writes :<sup>2</sup> “ Every case of copodyscinesia, without exception, has at one period or another of the disease some modification of normal sensation in the hand or arm. Usually the very first symptom that attracts the patient's attention is a sense of fatigue or tire in the hand or arm, which at first appears only after a considerable amount of work. . . . If the work is continued, the sensation increases. . . . When a subacute neuritis is present, as frequently occurs, all the symptoms common to that condition appear, viz. : pain over the various nerve-trunks and at the points of emergence of their branches, either spontaneous or only solicited on pressure ; areas of hyperæsthesia or anæsthesia ; a sense of itching or tingling or pricking in the arm or hand ; or a sense of numbness, causing the part to fall asleep.” In the following case, there was probably this latter condition of subacute neuritis :

Miss D., 23, single, is a carpet-pattern-setter at the Roxbury Carpet Works. On the 17th of May, 1886, she came to the nervous out-patient room at the Boston City Hospital, and gave the following history : Although always of a nervous temperament, she has been in good health until three or four weeks ago, when she began to have pain in both her hands, extending up to the arms to about the elbows. She had been working constantly before this attack, but knew of no injury or exposure to cold which might have caused her trouble. The pain was quite severe and at times rather sharp, although usually of a duller and more

<sup>1</sup> *Op. cit.*, p. 350.

<sup>2</sup> *Op. cit.*, p. 519.

fixed character. No special numbness or paræsthesia was noted, but the hands and arms felt lifeless and were easily fatigued. The trouble was worse upon the left side. On trying to set patterns, these symptoms were aggravated. Some trouble was experienced in doing the finer work at home, such as sewing, while coarser work, like sweeping, was easier. Any attempt to use the arms, however, caused some disturbance. She never noticed any spasm while at her work, and had no special difficulty in the co-ordination of motion, although, as I said, the motion itself aggravated the trouble. With the exception of this trouble, she felt quite well. There were no cerebral symptoms, beyond occasional headaches, such as she always had; and the thoracic, abdominal, and pelvic viscera performed their various functions in a normal manner.

The physical examination showed a girl below the ordinary height, extremely slender, and with particularly small arms. There were no signs of anæmia or of special emaciation, and she had noticed no diminution in the size of her arms since this trouble came on. The various movements of the hand and arm were performed without difficulty. On the ulnar sides of the hands, there was a faint diminution of sensation, and pressure over the nerve-trunks in the forearm caused some pain, especially over the left ulnar nerve near the elbow. Unfortunately, no electrical examination was made, and the dynamometer was not used to test the strength of the muscles; and the patient's failure to return at the time specified precluded any attempts at a more elaborate examination.

The character of the symptoms, their distribution, the absence of any other cause, like injury or exposure to cold, and the fact that the symptoms were aggravated by renewed attempts to work, at once led me to think that the cause of the trouble might be found in the patient's occupation. By the kindness of Mr. F. E. Simpson, the treasurer of the Roxbury Carpet Company, I was enabled to inspect the process of pattern-setting.

The girls employed to set patterns work in a large room, well-lighted and well-ventilated. The foreman at the factory informed me that the work was considered the most desirable in the factory, as the pay was good and the work was considered easy. Neither he nor my patient had ever heard of any similar trouble among the employes in that department. Two girls are employed on each pattern. They sit on either side of a table along which the threads for the pattern run. In an ordinary tapestry, or Brussels carpet, the pattern is formed by the longitudinal threads of the web—the warp. Each individual thread, which is dyed at various colors through its length, is wound on a spool, and these spools, in number equal to the threads of the warp, are placed, on a large table, on pegs on which they can turn. Each thread is then drawn off from the spool and runs under a clamp and along the table at which the two girls sit, so that the threads of the warp, several hundred in number, as they lie along this table, show somewhat indistinctly the pattern of the carpet. Having these threads thus be-

fore them, the two girls then proceed to get them into order, so that the pattern may be exact. With one hand on the spool side of the clamp, they pick up one thread after another, as necessary, and tighten or slacken it, in order to bring the colored parts into their proper places. The other hand is kept on the table arranging the threads in their proper order side by side, picking out those threads whose tension is too small or too great, and helping the other hand in regulating the tension. When the threads before them are all arranged, a second clamp, by the side of the first, is brought down, so as to make them fast, and they are then wound off on to a large cylinder, from which they are woven. In case of doubt, each girl has the pattern by her side, to aid her in arranging the threads. As the two girls face each other, it will, of course, be seen that they use different hands for regulating the tension and for arranging the threads, according to the side of the table on which they sit; but, in the case reported, I was unable to learn which hand was used to regulate the tension, and which to arrange the thread.

The process with the hand that regulates the tension is one that requires the use of the thumb and fingers in simple flexion and extension in picking up the threads, and also a slight lateral movement of the hand in pulling the thread one way or the other. The other hand is held in a more rigid position; all the fingers are employed, not only in flexion and extension, requiring the action of the lumbricales especially, but also in slight lateral movements, which would probably be performed by the interossei. The movements required are usually so slight that the muscles of the forearm, except the extensors and flexors of the fingers, would not often be called into use. This process particularly would seem to furnish the delicate movements of co-ordination and the state of muscular tension requisite for the production of the morbid phenomena. The process first described requires somewhat less complex movements, but still it might well give rise to such phenomena.

The case was not under observation long enough for careful study, but its evident ætiology makes it of interest from the fact that, as far as my knowledge goes, this special cause for a professional neurosis of co-ordination has never been reported before.

## REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILADELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF PHILIP LEIDY, M.D., PHYSICIAN-  
IN-CHIEF, AND CHARLES K. MILLS, M.D., CONSULTING PHYSICIAN.

### CASE VI.—*Paranoia.*

Reported by Dr. J. Chalmers Da Costa, Assistant Physician.

J. J.—, single, middle-aged, was admitted to the hospital August 5th, 1885. He was born in Ireland, and was a Protestant, with a good common-school education. He drinks, but never smokes nor chews.

His father is alive and well, and there was never any insanity in the family. His mother was subject to violent attacks of neuralgia. He has a sister and brothers alive and well.

When about 20 years of age, he states that he had lung trouble, which was shown by night sweats, spitting of blood, loss of flesh, and cough. These symptoms came on gradually, and lasted for several years; then he apparently recovered, and he has had no relapse. He had an attack of acute rheumatism in 1875.

On admission to the hospital, his friends stated that for a long time he had been drinking heavily, and that this attack, which was his first, began with mania a potu about a month before. He was evidently laboring under great fear; he refused food and was fed with a tube. He attacked all who approached him, cried and wrung his hands, and appeared to be in a frenzy of grief and terror. All his organs seemed to be normal, and no evidences of syphilis were found. Under hydrobromate of hyoscine he slept, and his bowels, which were constipated, were moved by calomel and magnesia sulphate.

August 6th.—His condition was unchanged; he was restless and agitated, and subject to hallucinations of a terrible kind; he attacked people and hurled maledictions at imaginary individuals. He was sleepless and refused food. His case was diagnosticated as acute melancholia.

August 20th.—He was somewhat better. He ate a little, but with evident suspicion, and said somebody wanted to kill him.

August 25th.—He was quieter and more rational, and ate better.

September 15th.—He was somewhat worse ; was singing and cursing.

September 20th.—He was decidedly better.

September 24th.—He left the hospital in the care of his friends.

He returned, of his own accord, in January, 1886, he said because of a bad memory. He complained that he would constantly forget recent events, although he remembered everything that occurred previous to his insanity. He was not nervous nor excited, and in other respects, besides this partial amnesia, seemed well. His memory gradually returned, until he even remembered many facts of his insanity.

Examination showed that his forehead was high, and his hair black streaked with gray. He had no marked defect of sight or hearing, and his expression was intelligent and good-natured. He had no signs of venereal disease, and his heart and lungs showed no lesion. His urine was normal. His motility was unimpaired, and his reflexes normal. He complained of feelings as of running water, fornication, etc., in different parts of his body. He had occasional hallucinations of touch and hearing, and fixed delusions of the systematized variety. These delusions related to the powers of spirits, and the fact that all disease was caused by evil, and all prosperity given by good spirits. He said they guided his hand and gave him knowledge by writing. He was not emotional, and his intellect, though much influenced by his delusion, was not in other respects injured. He argued fairly well upon his belief.

His own story was briefly as follows : " Some time before I came here, I began to get violent headaches, and to be wakeful at night, but this was not on account of pain. I felt depressed, but could not tell why. One night, when in bed, I heard a voice screaming, but I could not tell what it said. I heard it all night, and searched the room to find its cause. Then I began to hear it regularly at night, and soon after regularly in the daytime ; it got so bad that it kept me from working. After a time, this screaming was transformed into many voices, which said, ' they will kill you,' and I would ask who, but they would only repeat, ' they will kill you.' I struggled against these voices and did not at first believe them, but after a time I commenced to, and was much frightened. One day, I got wild and wandered over the city with the voices pursuing me. I tried to go home, but could not ; I could not go in, something held me back. I reached the house of a friend, who told me to go to bed there, and I did so. The voices were so bad that I put the bed against the door for safety. I heard hundreds of voices saying, ' John, they will kill you.' I got up and dressed and ran from the house and went to a police-station for safety. In the cell, the voices got worse and worse ; I became wild and then do not remember anything until I came here, or rather until I found myself here. The voices were still around, and they told me not to eat, for the food was poisoned ; and I did not, for I believed them. After a time, I determined to eat, and a bowl of

milk was offered me, and the voices said, 'John, it is nitro-glycerin;' but, as I could not live so, I drained it, and for hours was afraid of exploding. When I found I did not explode I knew the voices lied to me, and determined not to believe them any more. I had on several occasions seen ghosts, usually naked men with knives. Then the voices got weaker, and I saw no more ghosts. I wondered what the voices were, and it suddenly flashed on me that they were evil spirits. I think something told me this, and that I was pursued with devils and that I must drive them out. I determined to get rid of them by force of will, and have done so largely, although some are left, which I hear principally in the ticking of clocks and the creaking of my boots in walking. I determined not only to get rid of bad spirits, but to use good ones. I am doing so and now know that I shall live forever when I get them all out. I have cured myself of rheumatic pains, and have cleared my eyesight, being now able to read without glasses. I am writing the history of the world before the deluge, the good spirits guiding my pen, and the evil ones trying to hold it back. Some of these evil spirits I have captured by pressing down my pencil and quickly folding up the paper. I have thus caught a bottleful, and have in a bottle the veritable Satan. These spirits emit a foul air and cause disease by getting in different organs. When I finish my book, I will regenerate the world and cure all the sick, and then men will never be sick nor die."

The man who tells this interesting story is apparently sane now, except upon the subjects which centre round his delusion. His memory is good, and he is never violent nor depressed. He is good-natured, but very egotistical, because of his new acquirement, and pities me because I do not free myself from the spirits and live forever. His delusion is well systematized, and he has some reason for every possible objection to his theory, and argues tolerably well upon his absurd premises. Day by day he becomes more firm in his belief and more persistent in its defense. His intelligence is of a high order. He has travelled much in India and other countries. He may be considered an insane man of much ability. He stays in the hospital, of his own wish, to finish his book.

#### CASE VII.—*Acute Alcoholic Insanity.*

G. J.— was admitted to the hospital October 11th, 1886. He is white, married, 40 years of age, had a common-school education, and has been a sailor.

He has no children; he has two sisters alive and well, and four sisters dead, though from what cause he is ignorant. There has been no insanity in the family; and no consanguinity in the marriage of his father and mother.

When on shore, he drank largely and used tobacco, smoking and chewing. He was never sick, except with malaria. He never had a fit nor a sun-stroke, and never remembered having any head injury. He had no syphilis. The sight of his right eye was destroyed, some years ago, by the discharge of a shot, and he had a

cut on the cheek, which he got, two years ago, in a fight. On the 10th of September, about one month before his admission, he was stabbed in the abdomen, and badly injured, while drunk. He was taken to the hospital and lost much blood. He was not maniacal nor depressed before the injury. While going to the hospital in the ambulance, he began to see strange animals and demons. In the hospital, he was found to have a bad abdominal injury, which was dressed. He then passed into a state of wandering delirium, with marked delusions of persecution.

Examination showed he had slight bronchitis of the larger tubes. His pulse was very weak and rapid; his tongue was red and tremulous, and his appetite and digestion poor. His bowels were constipated, and his spleen markedly enlarged. He had a scar on his abdomen running from the umbilicus to the pubis, and one to the right of the linea alba in the middle. He had also a scar on his right leg, and one on his left cheek. He complained of great abdominal pain. He had unsystemized delusions of persecution, which were much more marked at night. He thought that the man who stabbed him was coming to kill him, and screamed with fear. He heard voices threatening him, and saw strange animals. He had no tremor of the extremities, and the symptoms remitted markedly.

He was given hyoscine for some nights, and this made him sleep. His bowels moved, and the pain in the abdomen soon disappeared (inunctions of unguentum hydrargyri were used). His temperature was normal. His pupils were dilated, and the eyes injected. He was given at night infusion of valerian, after the first few days. During the day, he was given peptonized milk and eggs, and, after a cool bath, was rubbed down with coarse towels. He was also given quinine in five-grain doses. After a week, the patient showed marked improvement; his delusions disappeared altogether during the day and became much milder at night, and in a few days more they also disappeared, and he slept without bromide.

He was paroled November 28th, 1886.

This man's case differed from ordinary acute mania in its ready response to treatment, and short duration, in its sudden access, and in absence of the rapid flow of ideas, simulating incoherence, which is frequently found, as also in not having any state of emotional exaltation. The case, however, was not considered to be one of delirium tremens; tremor, which is such a striking feature of this disease, being absent, except in the tongue. It was considered to be an acute alcoholic insanity, due to exhaustion from loss of blood, and shock, the patient's whole condition being an exhibition of weakness—weak pulse, dilated pupils, etc.

## Periscope.

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### ANATOMY OF THE NERVOUS SYSTEM.

**The Intra-Axial Course of the Auditory Tract.** By E. C. SPITZKA, M.D. (*New York Medical Journal*, Sept. 18th, 1886.)

The conclusions of this able article are based upon the anatomico-physiological method—a method with which most of us have become familiar through the works of Meynert, Gudden, and Forel. The method consists in a comparison of the relative development of certain nerve centres and nerve-tracts in animals having exalted or rudimentary special functions. Dr. Spitzka has studied the auditory tract of Cetaceans. These animals have rudimentary hind limbs, but a highly developed sense of hearing. Anatomically, we may therefore expect to find the pyramid (motor) tract poorly developed, but the auditory tract unusually well developed. These speculations are borne out by an examination of the cross-section of the brain isthmus immediately behind the post-optic lobes. This trans-section shows the following peculiarities: "There is no pyramid tract in the pons, and the middle part of the lemniscus appears to be absent. The brachium conjunctivum (Bindearm) appears crowded mesad by an enormous tract which corresponds to the lateral part of the human lemniscus. The continuation of the post. commiss. in the mesal division of the reticular field is seen . . . and of the inner division of the lemniscus nothing is seen except the very distinct bundle from the pes to the tegmentum. . . . The trapezium is relatively the largest in the animal kingdom. . . . The trapezium fibres can be seen massing into a longitudinal strand which . . . can be identified with a remarkably voluminous tract which occupies the situation of the lemniscus and passes into the posterior pair of the corpora quadrigemina." These hypertrophy experiments of nature show that the trapezium, the lateral (lower) part of the lemniscus, the posterior tubercles of the corpora quadrigemina, and the internal geniculate bodies are intimately related to the sense of hearing, corroborating the experiments of Baginsky and others, who found that these very parts would atrophy upon extirpation of the posterior or cochlear division of the auditory nerves. Spitzka concludes that the atrophy (experimental) and hypertrophy methods prove that the sound is transmitted by the following parts from the periphery to the cortical



centre: 1. Cochlea; 2. Post. division of eighth pair. 3. Trapezium of same side, then crossing to, 4. Part of lemniscus; 5. Post. pair of corpora quadrigemina; 6. Internal geniculate body; 7. Corona radiata; 8. Cortex of auditory field. B. S.

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**The Cortical Origin of the Fibres of the Anterior Commissure in Man.** By DR. N. POPOFF. (*Neurol. Centralblatt*, No. 22, 1886.)

The best authorities are at variance regarding the anatomical relations of these fibres. Some (Burdach, Meynert, Gratiolet, and others) traced the fibres of the anterior commissure into the temporal and occipital lobes; others claimed that they were spread throughout the whole area of the gyrus fornicatus. Ganser claimed that certain fibres of the anterior commissure originated in the olfactory bulbs. Popoff now publishes a case of softening in the occipito-temporal region. On the left side the softening involved the whole of the gyrus lingualis and the posterior portion of the inner margin of the gyrus fusiformis; on the right side the softened area was of a similar extent. On both sides the softening had penetrated as far as the lateral ventricles. The posterior surface of the right cerebellar hemisphere, and a considerable part of the superficial portion of the pulvinar were also softened. These foci of softening were due to a well-developed cylindrical aneurism of the basilar artery, all branches of this artery exhibiting marked atheromatous changes, and both *arteriæ occipitales* (Duret) having been blocked by large thrombi.

Microscopical examination of the brain-axis revealed degeneration of all the fibres of the posterior division of the anterior commissure. Gratiolet's visual fibres, which are near one focus of softening, were slightly affected, while the bundles of fibres from the olfactory bulbs to the anterior commissure exhibited no distinct changes. It must be noted, in addition, that the temporal lobes were not involved in the disease. From these facts the author concludes, 1. That the posterior division of the anterior commissure is mainly instrumental in connecting the two gyri linguales, but that it is extremely doubtful whether any considerable portion of these fibres take their origin in the temporal lobes; 2. That there is a negative proof that there are no fibres from the gyri linguales to the medulla oblongata; this being in accord with Charcot's views that focal lesions in the occipital lobe are not followed by secondary degeneration in the crura cerebri. Prof. Flechsig adds a note, reporting a similar case in corroboration of the above conclusions. B. S.

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PHYSIOLOGY OF THE NERVOUS SYSTEM.

**Recent Experiments on the Time-Sense, and on the Perception of Space.**

There have appeared in MIND during the year 1886 several

original researches that are of interest apart from their special physiological bearing. In a series of experiments on the Time-Sense, Mr. L. F. Stevens reaches conclusions directly opposed to those of Vierordt, Mack, and the pupils of Wundt. His method of experimenting, like that of Vierordt, consisted in impressing on the mind intervals of time by means of a metronome, and in reproducing the same after the metronome had been stopped. Vierordt found that the reproduced interval was larger than the standard when this was small; shorter when it was great; and between the two extremes was an interval which could be reproduced quite accurately. This "indifference-point" was not the same for different intervals, but varied between 1.5 and 3.5 seconds. Stevens, on the contrary, finds that there is an interval of time, the value of which varies between .53 and .87 sec., which can be reproduced with considerable accuracy; but with all other intervals an error is made which is *plus* for those above and *minus* for those below the so-called indifference-point. The results of several other experimenters agree with Stevens in fixing the indifference-point at about .71 sec., but otherwise are in accord with Vierordt.

Stevens attempts no explanation of the discrepancy, and leaves the question open, to be solved by future experiment. It seems fair to conclude, however, that we have within us some sort of a "time-keeper" that is set to measure intervals of about .71 sec., but which cannot be trusted to measure accurately intervals either longer or shorter.

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In his research on THE PERCEPTION OF SPACE BY DISPARATE SENSES, Dr. Jastrow enters a field almost wholly new, and obtains some interesting and valuable results. His problem has been to compare and determine the relative accuracy of judgments of linear extension by the *eye*, the *hand*, and the *arm*. The comparisons are made by reproducing judgments made (1) by judging lengths by fixating the eyes on them without motion of the eyeball; (2) by judging distances between thumb and forefinger ("span"); and (3) by judging distances by guiding a pencil over them with a free arm movement.

Each sense is in turn made a *receiving* sense and an *expressing* sense, according as it receives the linear impression, or expresses it by a length that is judged to be equal to the first.

Great ingenuity is shown in the construction of the apparatus for receiving the various impressions and expressing the judgments, and the results are important in showing the errors that we all make in judging linear extension.

If the *eye* is both receiving and expressing sense, small lengths will be underestimated, and large lengths exaggerated. If the *hand* is both receiving and expressing sense, small lengths will be exaggerated, and large lengths underestimated. If the *arm* is both receiving and expressing sense, all lengths will be exaggerated.

Taking, now, the cases where the receiving and expressing senses are different; if the eye is the expressing sense, all lengths are greatly underestimated; while if the hand is the expressing sense, all lengths are greatly exaggerated. The arm behaves differently toward each of the other senses, greatly exaggerating lengths whose impressions are received from the eye, and greatly underestimating lengths whose impressions are received from the hand.

In all cases the error decreases as the length (to be reproduced) increases. These results may be formulated by saying that if reproducing one sense by another results in an exaggeration (or underestimation), then reproducing the second sense by the first will result in an underestimation (or exaggeration) to about the same extent. The relative accuracy of the senses is found to be sight, span, motion.

The errors of the blind are like those of normal persons, but are smaller. In comparing the accuracy with which small differences of length can be recognized by blind and by seeing persons, the effect of practice in the use of the hand and the arm shows a marked superiority in favor of the blind. WILLIAM NOYES.

#### GENERAL PATHOLOGY OF NERVOUS SYSTEM.

**A Few Remarks on the Relation of Tabes and General Paresis to Syphilis.** By PROF. STRÜMPPELL. (*Neurolog. Centralbl.*, No. 19, 1886.)

Prof. Strümpell, who has rapidly advanced to the front rank of German neurologists, has written a short and suggestive, though rather bold, article on an almost trite subject.

First of all, Strümpell is convinced that there is an intimate causal relation between syphilis and tabes. In his cases of tabes, 61% were positively syphilitic, and if all doubtful cases were taken into account, 90% would be the more correct estimate. Regarding syphilis as a toxic infection, Strümpell urges that tabes is, in these cases, an example of the nervous sequelæ (*NERVOESE NACHKRANKHEITEN*) which so frequently follow upon infectious diseases, *e. g.*, diphtheria and typhoid fever. The author does not believe, by the way, that the primary toxic agent of diphtheria causes these secondary nervous troubles, but that a *CHEMICAL POISON* found in the body must be held responsible for these later affections. The author recognizes the intimate relation between tabes and general paresis, and is convinced that the latter also is the result of toxic poisoning of the system, which happens, in these cases, to select the brain rather than the cord or the peripheral nerves.

All this is in accord with the views which Prof. Strümpell has published at various times during the past few years. The toxic character of spinal cord and peripheral affections has certainly not been urged more frequently by any one than by the author of the paper under review. We have called this paper "suggestive;" so

it is, but it is somewhat vague at the same time. It is what the Germans term "genial," with a slight intimation that the hard facts are wanting to prove the case. In justice to the author, we add that he appears to be conscious of the defects of these brilliant generalizations.

B. S.

**Increased Tendon Reflexes in Disease of the Peripheral Nerves.** By Drs. STRUEMPELL AND MOEBIUS. (*Münchener Med. Wochenschrift*, No. 34, 1886.)

The authors relate two interesting cases of multiple neuritis, in which the deep reflexes of the upper and lower extremities were decidedly exaggerated. With the recovery, the reflexes returned to the normal state. They attribute the exaggeration of these deep reflexes not to the removal of inhibition, but to irritation of the ascending (sensory) portion of the reflex arcs. The painful points along the course of the affected nerves argue in favor of this view. These observations are of considerable importance, and add one more to the list of symptoms common to spinal cord and to peripheral lesions.

B. S.

**Hysterical Amaurosis.** *Quoted from BULETIN DE HYDROTHERAPIA, OF BARCELONA, in Lond. Med. Record*, June 15th, 1886.

A lady, aged 32, married, without children, of nervous temperament and weak constitution, had suffered from hysteria at an early age. While working with a sewing machine, having noticed that it stopped, stooped over to see the cause and was pricked by the needle in the left superciliary region. She was very frightened, and thought that she had put her eye out. An attack of hysterical epilepsy such as she had before supervened. On recovering, she found that she was totally blind. No lesions of the eye could be discovered; but though she was treated by several medical men, no improvement resulted. She was transferred to Barcelona for further advice, where, after four months hydro-therapeutic treatment, she completely recovered.

**Spasm of the Glottis and the Diaphragm.** (*GAZ. DEGLI OSPITALI*, October 20th, 1886.)

Last March, at the medical clinic of the University of Genoa, a man, married, aged 32, presented himself with the curious phenomena of a peculiar dyspnoea (respirations 90-100 per minute). There was no cyanosis or anything showing a lesion of the respiratory apparatus. There was an inspiratory shrinking of the epigastrium and the throat, and the laryngoscope demonstrated an inspiratory spasm of the larynx. This fact was not considered sufficient to account for the dyspnoea. Inspection showed a clonic spasm of the diaphragm which limited the inspiratory act. The diaphragmatic spasm gave rise to hiccough in the period of relative quiet, that is to say when the currents of air were sufficient to pro-

voke the characteristic sound, otherwise it was absent, though the spasm persisted.

The diagnosis was made of a neurosis of the respiratory centres, characterized by spasm of the glottis and the diaphragm, accompanied with spasm of the auxiliary muscles.

The patient, three days after, had recovered from the dyspnœa and the hiccough without any treatment. GRACE PECKHAM.

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**On Clonic Spasm of the Tongue.** By DR. GUISEPPE SEPILLI. (*Rivista Sperimentale de Freniatria e di Med. Leg.* Anno XI., Fasc. IV., 1886.)

This affection is a rare one. Erb relates a case of a child who, when it was necessary to push out the tongue, had a spasm which consisted of a very rapid movement of alternate pushing forward and retracting that member.

Berger describes two cases of idiopathic lingual spasm; in one the tongue came forward irresistibly with rhythmic contractions at the rate of fifty to sixty times per minute. The spasmodic attacks lasted half a minute, and were repeated several times in the day. The tongue took a rectilinear direction, and pointed came against the lips.

The other case was that of a man, 42 years, whose tongue from time to time, such as a few times a week, or a few times in the day, was protruded against his will from his mouth with great force. Remak cites the case of a man who had a rectilinear spasm of the tongue four or five times during a minute. After citing these cases, the writer adds his own, that of a woman who had psychical disturbance, contemporaneous with which she manifested a spasm of the tongue, which consisted of involuntary muscular contractions, rhythmical in character, which were limited to the right side. The tongue was drawn back and then pushed forward against the right dental arch. On the right side were also fibrillary contractions. The motions did not cease, but continued day and night with varying intensity at the rate of forty to fifty times or more per minute. The movement was produced by all the muscles of the right side of the tongue, the genio-glossus, hyoglossus, styloglossus, and lingual. In the right side of the neck there was a rhythmical swelling, which at first sight appeared like arterial pulsation, but was dependent upon the clonic contractions of the muscles. (The writer gives a myographic tracing.)

There was also from time to time a spontaneous clonic spasm of the right foot.

The spasm was relieved, but not cured, by bromide of potassium, three grammes daily, and by the faradism of the lingual muscles.

In conclusion, the writer speculates on the seat of the trouble, and decided that lingual spasms are functional, and may be considered a neurosis of the hypoglossus. G. P.

**On Paralysis Agitans.** By DR. L. JACOBSON. (*Berl. Klin. Wochenschr.*, August 23d, 1886.)

A short *resumé* of a dissertation in which the above subject is treated exhaustively. The main point of the article is simply this, that the muscles in a case of p. agitans are permanently in a condition of spastic tension or rigidity. This is a motor-irritation symptom. The author thinks the disease should be styled *spasmus agitans*, and not paralysis agitans. The tremor, the loss of muscular power, the peculiar facial expression, the pains, and the forced movements, all are to be attributed to the departure from the normal muscular tonus. B. S.

#### MENTAL PATHOLOGY.

**The Decrease of General Paralysis, and Climacteric Insanity in the Male.** DR. CLOUSTON. (*Annual Report of the Royal Edinburgh Asylum for 1885.*)

Dr. Clouston considers that there has been an actual diminution of mental disease in Edinburgh within the past five years, and suggests that this is due to the fact that these were "lean years." He takes as the test general paralysis, as this is the most marked of all forms of mental disease, is distinct from all others, and is more common than any other form directly produced by wrong habits and modes of life. "It is, in fact, that form that is least dependent on hereditary and unpreventable influences, and most dependent on controllable causes operating during the life of the individual." The years of 1873-77 were mostly years of plenty and of inflation of wages, and during that period Dr. Clouston had 115 cases of general paralysis out of 1,580, or 7.3% of the whole. In the last five years, 1880-85, years of dull trade and little money to squander, there were only 75 cases out of 1,667 admissions, or 4.5%. Such a fall in the prevalence of any important typical disease, comparing one period of five years with another, is a most striking medical fact, and Dr. Clouston raises the question whether it has resulted from lessened opportunity of drink and dissipation, or from a lessened excitement in the modes of life. This year he has only 11 cases, the lowest number he has ever known. He considers that the prevalence of general paralysis may fairly be taken as the index of the prevalence of all preventable insanity.

There is one variety of disease in elderly men that he considers is becoming more common. It is not the typical break-down in mind occurring after 70, but a very sudden break-down soon after 60, or even before that, in men who have worked hard and continuously, their work, perhaps, accompanied by excitement, strain, worry, or too high living. They are men with no hobbies, no country tastes, and unable to get regularly or to use rightly a yearly holiday. They seldom take note of the premonitory signs of brain wear, and they commonly, but not always, have some hereditary brain weakness that has hitherto been

latent. It is an acute premature old age. They lose flesh, become bloodless, are unable to sleep, find their work extremely irksome, cease to enjoy food, and become depressed and restless. Brain repair is impossible, as they are worn out. Clouston considers it climacteric insanity in the male, rather than senile insanity proper.

WILLIAM NOYES.

**Sobriety in the United States.**—J. J. Henley, Esq., local Government Inspector for Great Britain, who was recently sent to this country to inquire into the methods of dealing with the dependent classes in vogue here, has presented his report to Parliament, "by command of Her Majesty," upon the poor laws of certain of the United States. Mr. Henley's inquiries were confined largely to the older States of the Union, especially those of the middle and eastern portion, and, as a result of his labors, he concludes that we are, comparatively, a sober people. Referring to the close connection between the vice of drunkenness and the relative amount of pauperism in the United States, he says :

"I can hardly venture to express any opinion on this question from my short experience over a very limited area of that great country. But it is generally admitted that a considerable proportion of the population of prisons, lunatic asylums, and work-houses in this country (England) may ascribe its position directly or indirectly to this vice ; in some instances, the individuals alone being the victims, while in other cases, which I fear are too frequent, whole families become paupers or otherwise suffer.

"I was much impressed, and I confess greatly surprised, at the temperate habits of all persons who came within the range of my observations in the United States. In the railway cars, upon the steamers, at the hotel bars, and in the public streets, the contrast was in this respect most unfavorable to this country.

"During the period of my visit, the presidential election was everywhere causing the greatest excitement, processions representing the two parties patrolled the streets at night with bands, torches, fireworks, and other electioneering devices, but on no occasion did I observe the least indication of the excitement caused by drinking, or any approach to a drunken row. Could any town in England have borne the same strain with a similar result ?"

I mention these facts without comment or any attempt at an explanation of the causes which have produced these effects. I do not inquire whether they may be attributed to differences in the climate, the laws, or the institutions of the two countries, or to the existence of a strong public opinion in the United States. Upon this question, I do not pretend to judge. As to the accuracy of my observation, however, I may be permitted to call as a witness an Englishman, whose opinion will have great weight in this country. Dr. Bucknill, in his recent notes on American asylums, thus writes :

"I cannot quit Boston and its kindly and cultivated folk, who made my visit there so happy and interesting, without bidding it and them a word of affectionate farewell. They love the old country, though they are proud of having taken the first step to break away from it. I was with them when they celebrated the centenary of Lexington, and the remembrance had no bitterness. And if they are proud of the past, they may well be of the present, for that day I mixed with a great crowd of 150,000 New Englanders, the outpourings of the city and the gathering of the country into the villages of Lexington and Concord, and I sought for, but did not discover, one man the worse for drink. In all that vast crowd, which I may even fairly call a mob, for it was a most disorderly assembly, there were no drunkards nor roughs, and the only policemen to be seen were a few fat slouching fellows round the President, who could not, however, prevent the mob from stealing his train, so that he had to wait for another. If there had been the average English element of roughs and drunkards, such a crowd must have ended in a riot, for the people did just what they pleased without interference. They climbed on and jumped off the roofs of railway trains, clambered in at the car windows, rode on the cow catchers, surged over the roads and through the processions, and yet all in good temper, and stopping short of any positive mischief. All the day long I saw no quarrel or fight, heard no angry words even, there were no breaches of the people's peace, and the behavior of this curious crowd was to me the strongest revelation of what sobriety, culture, and self-respect may attain to in the deepest and thickest layers of the population."

CARLOS F. MACDONALD.

### **Mechanical Restraint in the Treatment of the Insane.**

—The Committee on Lunacy, in their report to the State Board of Public Charities, Pennsylvania, 1885, aptly say:

"Mechanical restraints have been abundantly proven to be worse than useless, having been abolished altogether in some hospitals, with the happiest results. They are usually irritating and degrading to the insane and complicate the treatment. But they cannot be dispensed with unless the corps of attendants is ample; and we have hesitated to insist on absolute abandonment in the hospitals of the State. They are, however, more and more, working their own way out. It is not many years since this was a common mode of treatment. That there is a rapid diminution in the hospitals of this State is evidenced by the fact that, at the close of this year, with a resident population of 4,482, there were but 26 patients under mild forms of mechanical restraint—a little more than one-half of one per cent. Last year there were 38, out of a population of 4,105."

An interesting feature of the lunacy committee's report is an appended paper upon "Progress and Tendencies in Care and Treatment of Insane During the Past Year," as shown by "the



last reports of the various hospitals and asylums of the country, and other sources," by Dr. John B. Chapin, the able physician-in-chief and superintendent of the Pennsylvania Hospital for the Insane. Dr. Chapin's long and successful career in the field of lunacy, together with his well-known progressive, yet conservative, tendencies, lends the weight of authority to what he here says respecting the increasing disuse of mechanical restraint in American hospitals for the insane; this he attributes, partly, to the greater care which is now given to the selection and training of attendants and others engaged to care for this class, and to whose immediate custody the inmates of our public asylums must of necessity be intrusted.

There is another and equally important factor in the solution of the non-restraint problem, of which, though not directly referred to by him, it may be assumed that Dr. Chapin is fully aware, namely, a growing disposition on the part of asylum superintendents, especially the younger ones, to disabuse their minds of the notion that a standard of perfection in the care and treatment of the insane has already been attained, and that nothing further is to be sought for in that direction. Unfortunately, there are yet a few "conservatives" among the fraternity of asylum superintendents, who persist in advocating and using restraint, apparently unable to appreciate the fact, of which every superintendent who has honestly tried it is convinced, namely, that the very conditions which formerly were regarded as necessitating the use of restraint, that is, noisy, violent, and destructive tendencies, diminish in a ratio directly proportionate to the disuse of such restraint. In fact, recent experience has amply demonstrated that *non-restraint has "come to stay";* and the time is at hand when the advocates of restraint, the number of which is, happily, rapidly diminishing, must be regarded as the exponents of an era in asylum management which is rapidly drifting into the back-ground. The reporter would earnestly commend the following extract from Dr. Chapin's valuable paper, to the thoughtful consideration of the few remaining representatives of the restraint school:

"It might be stated, as a proposition, that, as the quality of personal attendance improves, the record will show a diminished amount of mechanical restraint, so that the latter may be regarded as in some degree a gauge or measure of the former. Not a line has been written in defense of its use or advocating mechanical restraint as a humane measure. It is probable that the American practice and views will soon be in accord with the example set by Scotch and English medical superintendents in their admirable administration. As an evidence of what has been accomplished, it was recently announced that mechanical restraint had been wholly abolished in twelve asylums, and in a larger number it was so seldom resorted to that it had practically fallen into disuse. Dr. Bryce, of Alabama, in his report refers to the marked improvement in his asylum following the total abolition of mechanical restraint. There is less noise and violence, and a better rela-

tion prevails between attendants and patients. Dr. Hurd, of Michigan, has noticed that the growing disuse of restraint has changed the relation of patient and attendant. 'Unconsciously to himself, and almost imperceptibly, the attendant has become, not so much the keeper, but the friend, companion, and nurse of the patient.' Dr. Andrews, of Buffalo, notices the tendency in asylum treatment to increase of personal freedom until now a degree of individual liberty is generally allowed, which, at a period of time within the life of the present generation, would not have been deemed compatible with safety or even possible. Dr. Chase and Dr. Bennet, of Norristown, report that they have passed the year without the use of any restraint."

Singularly, yet doubtless with no intention of making invidious distinctions, Dr. Chapin, in particularizing asylums in which the use of mechanical restraint has been totally abolished, omits to mention the pioneer, American, non-restraint institution, namely, the State Asylum for Insane Criminals at Auburn, N. Y., where the use of restraining apparatus was practically discontinued in January, 1879, and in which there has been no instance of its use for a continuous period of nearly five years. In his annual report for 1884, the medical superintendent of the Auburn Asylum refers to the subject of restraint in the following language: "Tendencies to violence on the part of patients have greatly diminished, since the total and final abolition of mechanical restraint, two and a half years ago; while that which was known as the 'refractory' ward, under the system of chains, shackles, handcuffs, camisoles, muffs, wristlets, and 'crib' beds, formerly in vogue here, has gradually changed in character, until now it may justly be classed as a 'quiet' ward, although still occupied by the 'worst' and most troublesome cases.

"It would seem that the question of mechanical restraint in the treatment of the insane, regarding the propriety of which there has been so much discussion, and even bitter controversy, is rapidly settling itself, and that the disuse of restraint may reasonably be predicted, in the near future, in every well regulated hospital for the insane. In this asylum we no longer even think of using it. In fact, a majority of our present corps of attendants have but little or no idea of its mechanism, and would be at a loss to know how to apply it were it placed in their hands for that purpose. In the light of such experience, candor compels the admission that, whereas I formerly thought mechanical restraint almost a *sine qua non* in the treatment of a certain class of cases, and so advocated, I now not only regard it as unnecessary, but I sincerely believe that such cases may be managed far better and easier without it.

"It has been said, in defense of restraint, that American superintendents are obliged to resort to it because of a greater degree of turbulence manifested by the insane of this country, as compared with that of Great Britain, where, owing to an alleged national difference in temperament, insanity assumes a quieter

and less violent type ; that, given similar conditions as regards their mental manifestations, and any intelligent American superintendent would manage his patients without restraint. This seems plausible, and, formerly, I accepted it as furnishing a rational and satisfactory explanation of the difference in practice between the two countries in the matter of mechanical restraint ; but the marked change in the demeanor of patients which I have witnessed here, as a result of an impartial trial of the non-restraint system, has led me to regard the explanation as fallacious. Under the old system, as formerly practised here, could be seen to an extreme degree, the manifestations of violence, noise, and confusion, which have been characterized as the ' American type of insanity,' while under the present methods, the ordinary condition of all the wards is one of marked order and quietude, and it may now be said that the prevalent type of insanity here is similar to that described as existing in the British asylums. Our ' disturbed ' ward has faded out, so to speak, and its departure has been followed by a gradual extension of the means and methods of occupation, embracing agricultural labor, the manufacture and repair of all shoes and slippers used by the patients, of all clothing excepting stockings, our tailor shop being manned entirely by patients ; also the manufacture of tinware, as well as glazing, carpentry, painting, etc. With these facts before us, is not the inference a fair one that the ' quiet type of lunacy ' found in British asylums is *a result* rather than *a cause* of non-restraint ? Observations made during my visit abroad last year forced upon me the conviction that in this respect, at least, our English brethren are in advance of some of us on this side of the water. But already there are numerous indications of the commencement of a new era in the care and treatment of the insane in this country, and it may safely be predicted that the not distant future will witness a marked modification in the form of construction, organization, and methods of conducting our hospitals for the insane. Even now the most ardent advocates of the old system, still more or less prevalent, are, unconsciously, perhaps, gradually diminishing the amount of restraint used, and otherwise modifying their practice in accordance with the spirit of progress which now obtains."

CARLOS F. MACDONALD.

## Society Reports.

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### NEW YORK ACADEMY OF MEDICINE.

#### SECTION OF NEUROLOGY.

*Stated Meeting, October 8th, 1886.*

*The Chairman, L. PUTZEL, M.D., in the Chair.*

DR. JULIUS ALTHAUS, of London, a Corresponding Fellow of the Academy, read a paper on

#### SOME POINTS IN THE PATHOLOGY AND TREATMENT OF PARAPLEGIA DEPENDENT ON POTT'S DISEASE.<sup>1</sup>

DR. V. P. GIBNEY said that he had been much pleased with the clinical picture which the distinguished visitor had presented, and also with the admirable manner in which he had connected the various symptoms observed with the pathological conditions undoubtedly present. The case detailed was one of great interest, and he thought that the point in regard to the service rendered by the actual cautery was well taken. This agency, however, is not the only means at our command for securing benefit to the patient. He had seen a large number of similar cases perfectly relieved within a week by the simple application of a jury-mast. Some time ago Dr. Charles J. Poore, of this city, read a paper in which he related a number of cases in which he was able to report very good results in the treatment of Pott's disease by the actual cautery. Afterward, however, he was much disappointed in its use, as the results in other cases were entirely unsatisfactory.

The author of the paper had cited but a single case, and though in this particular instance the method was eminently satisfactory, he did not think this was sufficient to prove its applicability in general. Personally he believed the actual cautery to be objectionable, and particularly in the case of children. Although it is the present custom to apply it very lightly, as compared with the former practice, it is impossible to persuade patients that it will not hurt, and both the mothers and the children object very strongly to its use. Of course, this objection applies principally to private practice.

He thought, as Dr. Althaus had pointed out, that the case which improved belonged, not to the third stage, but to the neuralgic class of cases. He had a number of times applied the actual cautery in such instances, just as soon as any symptoms of

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<sup>1</sup> See p. 13.

paralysis showed themselves, but without securing any relief. When, however, he had stopped its use, and had resorted to suspension, and the application of a jacket, the patients got well. In these cases he was strongly in favor of giving large doses of iodide of potassium—say sixty, seventy, or eighty grains three times a day to a child. The objections to the use of the iodide in adults did not hold good in regard to children, with whom it almost always seems to agree perfectly, and who often grow fat upon it. With the iodide, and a jacket to support the spine, he had obtained better results than in any other way. The theoretical part of Dr. Althaus' paper he thought admirable.

DR. SACHS said he agreed with the author of the paper that interference with motion is much more common than with sensibility. With regard to the treatment, he had no doubt that the actual cautery is efficient in certain cases; and yet in the one related by Dr. Althaus, he was inclined to doubt whether the credit for the cure should be given to this procedure entirely. Thus, in addition, the patient was given complete rest and treated with cod-liver oil and other tonics. He had seen cases cured by complete rest alone. The same was true of many cases of chronic myelitis, and he called to mind particularly one case of post-puerperal paraplegia. He did not wish to discredit the treatment by the actual cautery, but it seemed to him that the other measures adopted were of considerable importance in bringing about the satisfactory result noted.

DR. L. PUTZEL said that he had had a few cases in which the results were similar to those obtained by Dr. Althaus in the case reported in the paper. In three instances the improvement was so rapid after the application of the actual cautery that no reasonable doubt could be entertained as to its efficient agency. In the larger number of cases, however, this procedure was entirely without beneficial results. As to large doses of iodide of potassium, as recommended by Dr. Gibney last year, he had found in his experience that these also were without effect. He agreed with the last speaker that, after all, time and rest are the most successful remedies. Good feeding, however, is a matter of great importance also.

With regard to the absence of anæsthesia in these cases, it seemed to him that the explanation presented in the paper was a very plausible one. It certainly seemed to be a fact that there is some quality about sensory fibre which makes it resist pressure much more efficiently than motor fibre.

DR. ALTHAUS, in closing the discussion said that, the good effect produced by very large doses of iodide of potassium, as claimed by Dr. Gibney and others, was a very interesting matter, and that on his return to England it would give him great pleasure to test practically the efficacy of the treatment. It had been assumed that the rest and other treatment, beside the application of the actual cautery, had had much to do with securing the good result noted in the case which he had narrated. But this he be-

lieved was not the fact. For four months in the provincial hospital, and for three months after, when she came under his own care, the patient had been treated with complete rest in bed, and in addition she had been taking cod-liver oil and iodide of potassium, the latter in thirty-grain doses three times a day. And yet no effect whatever was produced—the patient remained in precisely the same condition, without the slightest improvement. As soon, however, as the first application of the actual cautery was made (within less than ten days) a marked improvement in the paralysis commenced; and this rapidly became more and more marked under the repeated use of the cautery. Before resorting to this measure he had wished to give the rest treatment a thorough test, and as it was attended with no improvement, while from the first the improvement was marked under the use of the cautery, he thought that he was fully justified in attributing the cure to this particular method of treatment. There were, no doubt, cases in which the cautery had failed; but it seemed to him that in these instances the disease was probably more advanced. He inquired whether there was not some loss of sensibility, as well as motion in such cases? In conclusion, he said that his reason for bringing this subject before the Section was to induce surgeons to resort to the actual cautery at as early a period as possible.

DR. GRAEME M. HAMMOND read an elaborate paper on

ATHEIOSIS, WITH REMARKS ON ITS PATHOLOGY.<sup>1</sup>

DR. BIRDSALL said that in order to show that athetosis and allied clonic movements are dependent alone on lesions of the gray matter of the brain, it is necessary to prove that the internal capsule is not at all affected in the autopsies referred to. Athetosis so frequently follows hemiplegia, and especially in children, that it seems reasonable to suppose that the motor fibres are affected in it.

DR. PUTZEL mentioned two autopsies which he had seen, which bore upon the subject, and which went to confirm the position taken by the author of the paper. One was in a very marked case of chorea, of twenty years' standing, and the autopsy revealed pronounced a trophy of the central convolutions, with descending degeneration down the cord. The other was in a case of pre-hemiplegic chorea. He was glad to find that the author of the paper had not treated athetosis as a distinct disease, but as an affection allied to chorea, hemi-chorea, etc.

DR. HAMMOND explained that the greater number of the autopsies he had referred to were described with the greatest accuracy, many of them being accompanied with diagrams representing the seat of disease, and that in none of them were the motor fibres implicated in the slightest degree.

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<sup>1</sup> See Vol. XIII. of this Journal, p. 730.

THE  
Journal  
OF  
Nervous and Mental Disease.

Original Articles.

PROGRESSIVE PARALYSIS OF THE EXTERNAL OCULAR MUSCLES, OR OPHTHALMOPLEGIA EXTERNA.

By W. R. BIRDSALL, M.D.,

NEW YORK.

**D**URING the fall of 1883, a patient was referred by Prof. D. B. St. John Roosa from his clinic at the Manhattan Eye and Ear Hospital to my service in the Department for Nervous Diseases of the same institution.

The patient, a male, aged 18 years, by occupation a bookkeeper, and of American birth and parentage, presented a group of symptoms which I had never met with before, and which was also unique in Dr. Roosa's extensive experience. They consisted of paresis of all the external muscles of both eyes, namely, the levator, the recti, and the oblique muscles; with complete preservation of function in the internal muscles (iris and ciliary muscles), along with a normal fundus and normal vision, except defects due to an error of refraction, and without evidence of lesions in any other nerve tract.

His history is as follows: About two years ago, his friends first noticed a sleepy expression of his eyes before he was himself aware of the drooping of the lids. The right eye became affected first; it was not until about six months ago that the left became decidedly affected. For two months before admission, increase in this bilateral ptosis was rapid, but never quite complete. He also observed a gradually progressive impairment of power in rotation

of the eyeball in all directions, until his eyes had become almost completely fixed, so that he was obliged to depend entirely upon rotation of the head to widen his field of view. He had not observed any failure of vision, and had been able to finish his collegiate studies, and afterward to write at bookkeeping many hours a day without difficulty. He never observed diplopia. He says he is perfectly well, with the exception of his eye trouble, and appears to regard that as of little consequence. He had, during his student life, an occasional headache, but never since. He acknowledges masturbation in former years, but denies positively the possibility of his ever having had syphilis or any venereal disease. There is no history of former sickness, except that during childhood he was said to have "kidney disease," and at ninth year fell down-stairs, striking his head. Syphilitic or nervous disease cannot be elicited from his family history.

Examination on admission: Double ptosis, most marked on right side, both eyes two-thirds closed; both eyes act equally in rotation, but the range is limited in all directions; pupils react to light and to accommodative movements; accommodation normal; fundus normal. Dr. J. B. Emerson, who made repeated examinations of his vision, found an irregular astigmatism in the right eye, and myopia in the left. Facial muscles act normally, and react normally to the faradic and galvanic currents; tongue and palatal muscles normal; deglutition and speech normal; nothing abnormal in sensory or motor distribution of the fifth, or of any other cranial nerves, except the third, fourth, and sixth pairs. Motility and sensibility of the trunk and extremities found entirely normal after the most thorough tests. No cardiac or pulmonary lesion. Patient has a healthy appearance except that he is somewhat pallid; has cold, wet palms, and a somewhat embarrassed manner. His mental condition appears sound. None of the visible signs of locomotor ataxia, and no history of fulgurating pains.

The patient was treated for several weeks with galvanism (cathode on eye, anode on neck) without apparent effect, then for several weeks without electricity, but with tonic doses of *nux vomica*, also without apparent change in symptoms. He disappeared from the clinic for some weeks, and on his return was put upon increasing doses of potassium iodide (gr. xv., t. i. d., increasing gr. ij. per diem). About two weeks after beginning this treatment, the record states that patient appears to have slight increase in ptosis, but moves eyeballs somewhat better than before. Two weeks later, improvement in the movement of the eyes was more evident, particularly in the left eye; ptosis in the left eye being also less marked. Ninety grains of KI per dose had been reached at the time. No new symptoms had developed; vision, accommodation, reactions of iris, and the other functions of the body remaining, as at first, normal.

In attempting to explain the pathology of this, to me,



novel case, I reasoned as follows: We have impairment of function of the sixth, fourth, and part of the third nerves, on both sides. What is the nature and the seat of the lesion which will account for the involvement of these nerves, and, at the same time, allow the structure in the immediate vicinity to remain unimpaired? An intra-orbital lesion which involved the third nerve, would not permit the escape of the supply to the iris and ciliary muscle, nor would a lesion in any part of the trunk of this nerve, in its intracranial portion, account for it. We are, therefore, forced to conclude that the nuclei of origin are the parts involved. This view is strengthened from the fact that the affection is bilateral. Physiological and anatomical facts, notably the experiments of Gudden, and of Hensen and Voelcker, render plausible, if not demonstrable, the theory that the innervation of the sphincter iridis and of the ciliary muscle proceed from nuclei which, although in close proximity, are distinct from those giving origin to the remaining third nerve fibres which supply external ocular muscles. Our case would seem to be one in which the latter motor nuclei were involved, but not those for the internal ocular muscles. Assuming this, we are forced to another conclusion, namely, that the lesion cannot be a gross lesion. It is hardly possible to conceive of a tumor, a meningitis, an arteritis, or a focus of softening, affecting, bilaterally, nuclei so widely separated as the third, fourth, and sixth pair, and not at the same time involve the centres for the iris and ciliary muscle, the fifth or the seventh nerves, or other neural tracts, without we assume that several independent lesions exist, and have accidentally fallen upon the nuclei of a set of associated nerves. That such a marvel of chance should occur can hardly be entertained. It will be observed that all the muscles affected are associated to move the eyeball and remove the lid, constituting within themselves a physiological system distinct from the functions of the iris and ciliary muscles which are more intimately connected with true vision. The most rational hypothesis, then, is that we have a progressive degeneration of this system

primarily functional, but finally resulting in structural changes of a degenerative type similar to the processes in progressive muscular atrophy and in labio-glossopharyngeal paralysis. It is almost to be expected that this form of lesion should occasionally be found in these upper motor nuclei, as well as in the cord and medulla. While this view appears to be the most rational one, there are two others worthy of thought. Some parts of the nervous system resist the action of a pathological process in their vicinity, while neighboring parts have their functions interfered with thereby. It is barely possible that the oculo-motor centres might be affected by an inflammatory or neoplastic process along the ventricular surface near these nuclei which other nerve centres might possibly resist entirely or for a long period of time, and such a theory may account for cases in which affections of the motor nuclei are simply initiatory to a more widespread involvement of neighboring centres in rapid succession. Another still more plausible view is, that vascular changes have resulted in numerous minute hemorrhagic extravasations, those falling within motor nuclei inducing therein degenerative changes and more disastrous results than where they fall in conducting tracts or in sensory areas which are supposed to have a greater number of pathways open to impressions that usually traverse them. Such minute hemorrhages are frequently found after acute febrile diseases, also in nerve centres distant from some focus of disease in other regions of brain or cord. In my own cases, however, there is little to substantiate either of these views, while the slowly progressive course, symmetrical and systematic distribution of the paralysis, together with the absence of other signs of disease, point more conclusively to the view I have advanced.

I was obliged to confess to those to whom I broached this view that I was not aware of any autopsies that would confirm it, nor, in fact, of any published account of such cases. Not long after my examination of this case, however, I found in Ross' valuable text-book an ac-

count of a similar group of symptoms under the heading, Progressive Paralysis of the Ocular Muscles, which up to that time had escaped my notice, as well as the papers of von Graefe and Hutchinson on which his description was based, and on reading Hutchinson's paper, I found a similar view of the pathology of such cases expressed as that stated above in my own case.

Mr. Jonathan Hutchinson's paper was published in the "Medico-Chirurgical Transactions" for 1879. In one of his autopsies, made by Dr. Gowers, a lesion was found in the ocular motor nuclei resembling that found in progressive muscular atrophy.

Before referring again to Hutchinson's cases, the history of a second case will be presented which, by one of those strange coincidences in which rare events run in groups, came to my notice a few months after the first, for which I am indebted to Dr. F. M. Wilson, of Bridgeport, Conn., Assistant Surgeon to the Manhattan Eye and Ear Hospital, who recognized its identity with the former case, and kindly brought the gentleman, a private patient, for me to examine. His notes of observations upon the case are as follows :

J. E. D., aged 29, farmer, began in January, 1883, to see double. At first, the diplopia was intermittent, "would come on once or twice a day, but would last only a few minutes." About the last of April, this double vision became constant. Has now, June 2d, paresis of right internal rectus, R.V. =  $\frac{2}{3}$ °, L.V. =  $\frac{2}{3}$ °. Reads No. 1 (J.) with either eye up to six inches. About the middle of September, the lids began to droop, and in November the double vision ceased. Jan., 1884, there was marked ptosis, and the eyes were fixed so that they could not be moved in any direction. R.V. =  $\frac{2}{3}$ °, L.V. =  $\frac{2}{3}$ °. No. 1 (J.) read at six inches. No other symptoms. He denies all venereal trouble, and states that he "has not been sick a day since he was fourteen years old." Potassium iodide was ordered, gr. x. t. i. d., increasing gr. i. per day. Faradism to the eyes. June 9th, 1884; he has had the faradic current almost every day since January. The potassium iodide was gradually increased up to gr. lxxx. t. i. d., which he is now taking. There has been a slow, gradual improvement. He can now move his eyes horizontally through an arc of about forty-five degrees, and up and down through an arc of about thirty degrees. There has been some improvement in the ptosis, but not much; at no time since November, 1883, has he had diplopia. Up to the

1st of May, his eyes moved slowly and with effort. They now move quickly to the limit, and no amount of effort carries them any farther.

As a result of my examination, no defect was found in any other nerve distribution than the sixth, fourth, and external muscular supply of the third nerves on both sides, except a suspicion that the upper or orbital group of the facial muscles responded to electrical excitation not quite as actively as upon the opposite side; the difference was so slight, however, that not much stress could be placed upon it. Both cases here reported were exhibited by Dr. Roosa at meetings of the New York Ophthalmological Society, and, I am informed, failed to elicit any new cases, except one which Dr. Kipp, of Newark, said he had observed. On writing to Dr. Kipp, asking if his case corresponded to those here reported, in exhibiting paralysis of the external muscles of both eyes only, he was unable to give me exact notes, but thought his case was the same in character.

To Mr. Hutchinson we are indebted for that convenient classification of paralytic affections of the eye, or ophthalmoplegia, into those affecting the extrinsic or external muscles of the eye (*ophthalmoplegia externa*), and those affecting the intrinsic or internal muscles of the eye, namely, the iris and ciliary muscle (*ophthalmoplegia interna*), the latter group being divided into *cycloplegia*, or paralysis of the ciliary muscles, and *iridoplegia*, or paralysis of the radiating and circular fibres of the iris, again subdivided into *myosis paralytica* and *mydriasis paralytica*.

Under the title "Ophthalmoplegia Externa, or Symmetrical Immobility (Partial) of the Eyes, with Ptosis," Mr. Hutchinson reports seventeen cases, to which he refers, as follows: "They are characterized by a very peculiar group of symptoms. Drooping of the eyelids, so as to give to the face a half-asleep expression, is usually first, and it is soon accompanied by weakness of all the muscles attached to the eyeball, so that the movements of the latter become much restricted, or even wholly lost. The con-

dition is usually bilateral, though it is not always in exactly in the same degree on the two sides. Its symmetry probably denotes that it is of cerebral origin. It by no means always happens that all the ocular muscles are alike affected, or that they are attacked symmetrically, still it is a very marked feature of the malady that the muscles fall in groups, and not singly. Non-symmetrical paralysis of single ocular muscles is, of course, very common, especially in connection with syphilis and locomotor ataxia, but such cases are to be distinguished from those which I am now describing: first, by the fact of non-symmetry; secondly, by the early completeness of the paralysis; and thirdly, by the ease with which very frequently they are cured. In the majority of them there is, perhaps, good reason to suspect that a gumma in the nerve track is the cause. In the symmetrical cases now under consideration, however, the changes probably begin centrally; they are usually slow in progress, and are often difficult of relief. They agree with the single nerve cases, in that they occur chiefly in those who have had syphilis. Although I have ventured to speak of immobility of the eyeballs, I by no means wish to imply that it is usually complete; on the contrary, incompleteness in the degree of paralysis is almost as marked a feature as is the tendency to affect many muscles at the same time. Although the eyelids droop, there is seldom complete ptosis; great limitation of the range of motion of the eyelids is more common than fixation. The degree, however, varies with the stage, and at a later period, the paralysis may be absolute. The third, fourth, and sixth nerves are, of course, those which are involved, but not infrequently in the early stage one or more of these may wholly escape. Occasionally, the optic nerve itself is involved, and sight is lost. I am making these statements from a limited number of cases, for the condition is but seldom seen."

Concerning their probable pathology, he says: "The cases in question are probably closely allied in nature to what is known as progressive muscular atrophy, their peculiar features being that only one special set of muscles

(or rather nerves) is at first attacked. We have probably in them a very close parallel to the so-called bulbar paralysis, the labio-glosso-pharyngeal paralysis of Duchenne. In it, as in ophthalmoplegia externa, central degenerative changes occur, and the result is the paralysis of a set of associated muscles. It may be plausibly conjectured that the initial lesion is inflammatory of the nuclei of the affected nerves, which, in a slowly serpiginous manner, creeps from place to place along certain definite anatomical paths. Within certain limits, its directness of spreading and its progressive tendency may vary in different cases, but, speaking generally, the cases are remarkably the same in their features. In exceptional instances, definite symptoms of locomotor ataxia are present, and in others still more rare, the fifth nerves or the seventh, or even the eighth may be involved."

Curiously enough, although Mr. Hutchinson calls these, cases of ophthalmoplegia externa; of his seventeen cases, in only three is it positively stated that both the iris and ciliary muscles were not involved; in a fourth, they were normal on one side and involved on the other. He admits, however, that the ophthalmoplegia interna is "often, indeed, usually associated with symmetrical ophthalmoplegia externa." While the involvement of the internal muscles does not exclude the possibility of the lesion being that which the Hutchinsonian pathological theory involves, still we cannot be as certain that a neuritis, consequent upon basal meningitis, ependemitis, or neoplasms, may not have been the cause, particularly as in several of his cases other cranial nerves and nerve-tracts were involved; in some cases, the optic, producing atrophy; in others involvement of the fifth, and also involvement of the motor or sensory functions of the extremities and trunk. It is in the closely-defined character of the symptoms, therefore, involving an associated set of functions only, that the two cases whose histories I have reported are of such interest and value as supporting Hutchinson's view. We cannot tell what new involvements these cases might have developed if left to themselves, or may yet exhibit not-

withstanding treatment. The extension to other motor nuclei in the central gray matter, either above or below these first involved, would not be inconsistent with the theory of systematic degeneration, in fact goes to confirm Hutchinson's view that these cases form a part of the clinical history of progressive muscular atrophy.

Paresis of any of the external ocular muscles, pointed out by Duchenne as one of the earliest signs of *tabes dorsalis*, naturally comes to mind in the consideration of such cases. Of Hutchinson's cases, seven out of the seventeen presented some symptoms suggestive of this disease. Unfortunately, the pupillary phenomena, discovered by Argyle Robertson, of reflex immobility to light, with preserved mobility to accommodative reflexes, had not been described when Hutchinson's paper was written. Its frequency and importance as an early sign of *tabes dorsalis* is now well recognized. In a number of Hutchinson's cases, the sphincter pupillæ were parietic; in others, fixed and of medium size, but whether to light alone is not stated; the involvement of the optic nerve and the ciliary muscles and the external muscles introduces complications which render it difficult to estimate the value of the findings. In the cases which I have reported, however, we have no pupillary impairment and no optic atrophy, the pupillary reflexes are preserved, there is no history of the characteristic pains of *tabes dorsalis*, and no history of syphilis. Yet it is possible that time may show a development of these signs.

It would consume too much time to analyze all of Hutchinson's cases, the greater number of which, as already shown, are more complicated than those reported in this paper. The case with an autopsy, however, demands notice, as it is the one on which he founds the pathology of the disease, grouping it with bulbar paralysis of the progressive muscular atrophy type, while to my own mind it presents more of the clinical feature of a case of *tabes dorsalis*. It is but fair to state that the author himself says that this particular case "in part resembles locomotor ataxia, and in part progressive muscular atrophy."

Male, æt. 48, gardener; in 1869 had slight paresis of right sixth nerve (slight convergence and diplopia); right pupil rather larger than the other; near vision defective from weak accommodation; distant vision nearly perfect. Four years later, in right eye barely perception of light, left eye scarcely reads No. 20; could not abduct either eye; right eye habitually crossed inwards. No positive paralysis of other ocular muscles, but all acted feebly; sleepy look from drooping of lids, but can lift lids with effort. Left interni weaker than rest. Optic disk very pale, arteries and veins much reduced in size. He had been liable to attacks of severe pain in his forehead, sometimes for a week at a time or more. Cramp in the legs for seven or eight years at night; bowels somewhat constipated. At this time the pupils did not react in the least; left, of medium size; right, larger; no habitual headache. Mercury pushed to pyalism without definite benefit. In 1874, pupils motionless, external recti paralyzed, all others imperfect. Bowels very costive, sensation of tightness around abdomen like a strap; numbness of skin over abdomen and on face, slight on hands, a little on the feet. Later, aggravation of symptoms. He became absolutely blind; had a sort of "choking fit." Still later, his extremities failed him, were usually "icy cold," and he became bed-ridden, suffered dreadful pains in his head, and was frequently out of his mind; could eat, speak, and swallow well. He died in this condition seven years after the commencement of the symptoms. No history of syphilis, but eldest child, æt. 20, had notched teeth, and had had a most characteristic condition of syphilitic keratitis.

Autopsy, in which the brain only was obtained. Examined by Dr. Gowers.

Brain somewhat softened from commencing decomposition, cranial nerves examined in fresh state, pons and medulla after hardening. Nothing abnormal in convolutions or in corpora striata; hyperæmic patches in left lenticular nucleus; posterior tubercle of thalamus a little softer and smaller than normal; olfactory nerves normal; optic nerve and chiasma uniformly gray, fair consistence, optic tracts also gray; whitish striations in pons. Microscopical examination of nerves and tracts showed fat globules and degenerating fibres, but also a large number of healthy fibres. Third nerves smaller than natural, gray and translucent, very few healthy fibres, some fibres undergoing degeneration; numerous connective-tissue nuclei. In crura cerebri the tracts of fibres of origin are indicated by numerous connective-tissue fibres; scarcely any nerve fibres could be seen. In their nuclei beneath nates, disappearance of almost all of the multipolar nerve cells, two or three only to be seen in each section. A few cells of some size, but without processes, were seen, also very abundant minute angular cells not larger than connective-tissue nuclei.

Fourth nerve: No trace to be seen; their nuclei beneath testes presented a similar degeneration to that found in the third nerve nuclei.



Fifth nerve: Upper fibres of root appeared healthy, but lower fibres had a gray appearance, granular degeneration and segmentation; within pons little recognizable alteration in fibres of nerve; nuclei for most part normal; nuclei of motor roots of fifth normal.

Sixth nerve: Reduced to fine gray threads, with scarcely a nerve fibre to be seen, also in tracts of origin within pons. The so-called conjoined nuclei presented general degeneration, most of the large cells had disappeared, and only granules, nuclei, and small angular cells remained.

Facial nerves: Perfectly normal in the trunks and roots of origin within pons; nerves and nuclei of the auditory, glosso-pharyngeal, pneumogastric, and hypoglossus normal; peri-vascular erosions found throughout médulla, pons, and corpora quadrigemina large and numerous. In the lower part of floor of the fourth ventricle some areas of disintegration in gray substance just beneath the lining membrane, and the surface, partly from this cause, is more or less irregular. One such area appeared to have been caused by a small hemorrhage. No indication was found of pressure upon nerves, or of any acute changes in their nuclei. Dr. Gowers also remarks that the disintegration and connective-tissue changes are those continually met with in the gray matter of the cord in progressive muscular atrophy in the nerves and their nuclei of origin.

As the cord was not obtained, we cannot determine the question whether sclerosis of the posterior columns of the cord was not also present. Buzzard has called attention to these cases of symmetrical involvement of the ocular muscles as a part of the clinical history of tabes dorsalis. Thus these two great systemic degenerative diseases of the nervous system, the one affecting principally the motor nuclei of the central gray column, the other involving simply the sensory tracts, would seem to merge into each other in many cases.

I have not attempted to review the great mass of neurological and ophthalmological literature pertaining to this subject with any degree of thoroughness. Similar cases are no doubt hidden under names which give the searcher no clue to their existence. It may be mentioned, however, that von Graefe, in 1869, reported a case of symmetrical paralysis of the external ocular muscles only. The attack was sudden, of comparatively brief duration, and terminated in recovery. It is of interest, in that he attributed it to a basal meningitis.

H. Breusgin has reported the following remarkably interesting case:

A female, æt. 25, without previous illness, developed in September, 1875, diplopia from paresis of the right sixth nerves. In the spring of 1876, bilateral ptosis and slow but regularly progressive paresis of all the external ocular muscles of the left eye occurred, with perfect preservation of vision, power of accommodation and pupillary reaction, and a normal fundus. The lids could not be completely closed, indicating involvement of the orbicularis palpebrarum. In 1878, the right eye became affected; in 1879, both bulbi were immovable. The speech began to be decidedly affected, while the lips and tongue were freely active, and the palate exhibited nothing abnormal to sight. Yet the patient had a decidedly nasal speech, and the consonants *p* and *b* could not be pronounced distinctly. Difficulty in deglutition also followed, and distinct emaciation followed, which finally became very great; speech becoming unintelligible, and deglutition performed only with the greatest difficulty, death at last resulting from syncope. Up to the last moment, the iris and ciliary muscles remained normal. There was no autopsy.

The author groups his case with those of progressive bulbar paralysis, and the lesion, he believes, was some destructive process, be it a neoplasm or a focus of softening in the floor of the fourth ventricle and aqueduct of Sylvius, destroying the nuclei of the affected nerves: namely, the third, fourth, sixth nerves, and knee of the facial whose first branch was affected. He explains the escape of the third nerve supply to the internal ocular muscles on the supposition that nuclei for these muscles are situated more anterior (cephalad) than the other nuclei, which view he bases on Hensen and Voelcker's experiments to that effect. He also refers to Alfred Graefe's view that some anomalous anatomical variation may be present in those cases in which the iris and ciliary muscle escape. The author cites three similar cases reported by Foerster, whose paper was inaccessible to me (*Med. Soc., Breslau, 1878*).

*Note.*—The publication of the preceding paper has been purposely delayed since its presentation to the American Neurological Association, in order to watch the progress of the cases reported. I am able to say that now, after an interval of two years and a half, the condition of both

cases remains practically unchanged, but slight improvement has occurred, and what is of the utmost interest, there are no new symptoms or indications of the extension of the disease to other nerve tracts; in this respect these cases seem to be unique. The manuscript has been left as written in June, 1884, which accounts for the absence of reference to recent literature.

# ASSOCIATE EXTERNAL OPHTHALMOPLEGIA OR UNCOMPLICATED PARALYSIS OF THE EXTERNAL MUSCLES OF BOTH EYES.<sup>1</sup>

BY DR. W. F. MITTENDORF,

NEW YORK.

**P**ARALYTIC affections of the muscles of the eye are becoming daily of more importance, whether occurring separately or in conjunction with other affections, because they are of such great help in locating lesions of the brain. Thanks to the labors of Hensen and Voelcker in 1878, the location of the nuclei of the principal motor nerves of the eye are known to us; and many features of paralytic affections of these nerves, that before appeared mysterious, are now more readily understood, and I hope the time will not be distant when the ultimate origin of these nerves can be definitely traced to the gray matter of the cortex. At present there is great confusion in this respect, as some observers, like Munk and Carville, locate the centre of motion for the eye in the *angulus gyrus*; others, like Hensen and Voelcker,<sup>2</sup> look for it in the temporal lobe, and Hitzig in the convolutions of the frontal lobe.

There is, however, no doubt that in many instances paralysis of the ocular muscles is caused by lesions in the nuclei which are found in the floor of the third and fourth ventricles and in the *iter* connecting them. Of the greatest interest are those lesions of the third or motor *oculi* nerve which result in paralysis of a portion of this complicated nerve, leaving other parts of it intact. The most striking

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<sup>1</sup> There has been some delay in the reproduction of illustrations accompanying this article. They will appear in the next number of the *Journal*. — EDIT.

<sup>2</sup> v. Graefe's *Archiv*, xxiv., 1, pagina 1, 1878.

of these are those cases in which only the internal muscles of the eye, supplied by the third nerve, the sphincter of the iris, and the ciliary muscles are affected, or still more so those in which only the external muscles of the eye supplied by the third or motor nerve are implicated. These cases go to prove that there must be separate nuclei for the different parts of the nerve going to the separate muscles, and that there is a distinct origin for those fibres going to the internal, and those going to the external muscle.

It must not be forgotten that there are two eye muscles which are not supplied by the third nerve, but that the nerves going to them arise from nuclei in close proximity to those parts of the third nerve which supply the external ocular muscles. These different nuclei are separated from each other in such a way that one of them may become the seat of disease without the neighboring nuclei being affected, but, on the other hand, lesions may readily extend from one to the other until quite a number of them are diseased. This occurs, in fact, often enough during the growth of tumors developing in these regions, and as new nuclei become involved, new additions to an existing paralysis will be observed, until eventually all the muscles of one or even of both eyes may become paralyzed in this manner. This is by no means so very rare in persons affected with syphilis or tumors of the brain.

Of greater scarcity, however, are those cases where, in a comparatively short space of time, all the muscles of both eyes, with the exception of those in the interior of the eyeball, become paralyzed. The first one of this kind was described by the illustrious v. Graefe, in February, 1868. In his clear and concise description of the case, all the characteristic symptoms of this rare affection are admirably presented, and it was he who gave this affection the name of *ophthalmoplegia externa*.

The English literature has no mention of the affection until J. Hutchinson, in 1879, only seven years ago, reports a number of cases, seventeen in all, of which, however, only a limited number, three, belong to this class. In his

usual clear way, he describes the different forms of ocular paralysis; he speaks first of an ophthalmoplegia interna and externa, but the particular form, the plain, uncomplicated ophthalmoplegia externa of both eyes, is hardly sufficiently appreciated, nor is its pathology clearly understood. Great credit is due to Förster, who was the first to locate the seat of the lesion in the floor of the aqueductus Sylvii, which was apparently proven by an autopsy in one of Hutchinson's cases. I say apparently, because this was in a case complicated by other paralytic lesions, especially that of the internal muscles of the eye. Perinaud was the first to point out this difference in the lesions of ophthalmoplegia externa and interna, and in 1884, Dr. Birdsall, of New York, in reporting two cases of this kind, emphasizes that the lesion must affect the posterior nuclei of the third and those of the fourth and sixth nerve.

Of the greatest importance, however, is the admirable paper on the subject by Prof. Mauthner, of Vienna. In reporting three cases of this kind, he proves in a very able manner the great probability of the lesion being in the region pointed out by Förster and Birdsall; he thinks that a poli-encephalitis, similar to a disease of the gray ant. horns of the spine, which Kussmaul has named poliomyelitis, is the most probable condition to give rise to the disease.

What evidence have we got to prove the correctness of this theory?

Of post-mortem examinations, we have only four that belong to this class. Of these, the first one is that of a case of A. v. Graefe. But this patient, who had syphilis, and suffered from drowsiness and headache, and about whose pupils and accommodation nothing is known, and who eventually died of bulbar paralysis, presented nothing abnormal; at least no evidence of a gross lesion was found.

The second one is that of Goyet; he found hyperæmia and partial softening involving the iter and the floor of the fourth ventricle; the floor of the third ventricle, and consequently the nuclei of accommodation and of the sphincter being normal; but *this was likewise a case compli-*

cated with drowsiness, general atony of the muscles, and hemiplegia of the right side.

The third is that by Gowers, of one of Hutchinson's cases. This case is one of syphilis in which the *internal muscles of the eye were affected at the same time*. Here a degeneration of the nuclei similar to that observed in the spinal nerves in progressive muscular atrophy was found.

The fourth one is that by Bristowe, of a case of F. Warner. This history is of such interest that I shall report it here. Marion H., 25 years, began to have scanty menstruation and Graves' disease in 1877. 1880 she had diplopia, and a little later the eyes became fixed, and in 1881 dyspnœa and vomiting troubled her. Dr. Warner reported her case in 1882. Shortly after this she came under Bristowe's care. She had now epileptic fits, followed by paralysis and rigidity of the left leg and arm, with increase of temperature. No optic neuritis; nor was the iris or the accommodation impaired in the least. A little later she died of bronchitis. The result of the post mortem examination is as follows: *No visible* changes of any part of the cord or brain or intracranial tissue could be detected. In the hardened specimens, no morbid change could be discovered on microscopical examination, except some small, pale-yellow patches, not differing, however, from the normal structure, except by their color. These spots were found in the gray as well as in the white matter of the brain, and appeared to be local areas of anæmia, as they are sometimes met with in otherwise normal brains. They were found especially, 1st, in the cortex of the third left transverse frontal convolution; 2d, in the anterior extremity of the left lenticular nucleus; 3d, in the internal capsule of the adjoining posterior portion of the left lenticular nucleus. Microscopic examination showed the nuclei of the sixth and seventh nerve, as well as the corpora quadrigemina, normal. The nuclei of the third nerve were not examined by mistake. The left third frontal convolution itself, as well as the rest of the brain, as well as the medulla, the spinal cord, as well as the sympathetic nerves, were healthy.—It is unfortunate

that the third nerve nuclei were not examined, but as those of the abducens were perfectly normal, these were probably so.

Here we have four autopsies, and in every one of them the ophthalmoplegia externa had been complicated with other brain or nervous symptoms ; they are therefore not conclusive in regard to the pathological lesion of this rare disease. In fact, there has not been so far a single post-mortem evidence of the lesion, which gives rise to uncomplicated ophthalmoplegia externa of both eyes. The evidence so far seems to point to a nuclear change, but whether this is due to anæmia or hyperæmia, or to a special malnutrition, or whether we have to look for a lesion of the gray matter of the cortex representing a centre for the movements of the eye, the existence of which is even doubtful at the present time, the future will have to decide. So much is apparent that the lesion causing this disease cannot be fascicular, nor basal, nor orbital. The knowledge of the disease is, however, of such recent date, and the affection itself is so rare, that this fault seems to be excusable. In fact, there have been only about thirty cases of this kind recorded.

A. v. Graefe	reported	3 cases.
M. Benedict	"	1 case.
C. Schroeder	"	1 "
Alfred Graefe	"	3 cases.
Goyet	"	2 "
Camuset	"	1 case.
E. Raehlmann	"	1 "
J. Hutchinson	"	3 cases.
Buzzard	"	2 "
Lichtheim	"	1 case.
Foerster	"	3 cases.
Mauthner	"	3 "
Uthoff	"	3 "
F. Warner	"	1 case.
J. Hook	"	1 "
J. Bristowe	"	1 "
W. R. Birdsall	"	2 cases.



Mittendorf	reported	1 case.
Bull	"	2 cases.
Strümpell	"	1 case.

Going carefully over all these cases, I found that eleven were complicated by paralytic affections of other muscles or with marked nervous lesions, and only twenty-two were of the typical form, and occurring in perfectly healthy individuals.

*Time of the Attack.*—This appears to be especially between the ages of fifteen and forty years. Sometimes the affection appears to be congenital, and it may come on as late as fifty-five or sixty.

*Sex.*—This does not seem to make much difference, a number of the cases occurring in young girls.

*The duration* seems to be indefinite. Mauthner reports one case that remained unchanged for twenty years. Alfred Graefe, one of fifteen years' standing; A. v. Graefe, one that lasted very long, and the case I wish to call your attention to has remained practically unchanged for three years. Strümpell mentions another of twenty-five years' standing.<sup>1</sup>

The etiology of the affection seems to be as uncertain as the exact seat of its lesion. It appears to attack persons in perfect health; but in many of the cases a specific history seems to exist. Traumatism may give rise to it, and it may be congenital.

*The mode of attack* is, as a rule, slow; in most of the cases the motor oculi suffer first; in some the external recti are first affected. It takes generally several months before all the muscles are affected.

*Complications* do, as a rule, not exist, and the patients may enjoy perfectly good health otherwise. It has been observed that during the existence of the trouble, epileptic attacks come on, that bulbar paralysis, and sometimes general progressive muscular atrophy followed. Locomotor ataxia has been observed in some cases. In v. Graefe's case, bulbar paralysis came on five years after the beginning, and in Bristowe's case it came late, but the time is not stated.

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<sup>1</sup> Dr. Birdsall's case has remained unchanged for at least two years.—ED.

*The prognosis* is undoubtedly not very good, as far as perfect recovery is concerned, but it is likewise not apt to endanger the life of the patient. One recovery is mentioned by Mauthner, in a girl five years old, where the paralysis disappeared in two months without treatment.

Partial restoration of some of the muscles has been observed more frequently. The muscles which seemed most apt to recover some power were the levators of the upper lid, the superior oblique, the inferior recti, and lastly the internal recti, and in very rare instances the superior recti and the inferior obliques.

*The clinical history* of the cases is simple. If the levator muscles become affected early, and to a considerable extent, the dropping of the upper lid will interfere with the sight of the patient, and this will bring him to seek medical advice early; if the other muscles become affected first and if the disease progresses slowly, the patient may be inconvenienced by it so little that he does not apply for treatment until the disease has advanced considerably. Diplopia does not exist, as a rule, nor is there much exophthalmos which might attract the attention of the patient. This is to be explained by the fact that the oblique muscles, which, acting as antagonists to the externi, pull the eye forward in those cases where these latter alone are paralyzed, are involved in the process, and it is only in the case of Lichtheim where this symptom is mentioned. In Warner's case, the proptosis was thought to be due to Graves' disease. Squint is sometimes observed; it is, as a rule, divergent and of moderate extent. It is usually due to the effort to exclude one eye from vision, especially for near objects. Monocular vision is apt to take the place of binocular vision, even for the distance, if the internal recti are much affected. The eyes being fixed, the patient is not able to look sideways without moving his head; he is apt to pass his friends on the street without recognizing them, nor is he able to write or read with comfort, because he can use only one eye at a time, and has to hold his head in such a way as to favor this eye; nor is he able to read without moving his head or the book; the vision of

the patient remaining otherwise perfectly good, this may be the first thing to call his attention to his eyes. After some time, the patient becomes accustomed to the condition of his eyes, is not inconvenienced by it, nor is the condition likely to attract the attention of his friends if his eyes are deeply set; but in persons with very prominent eyes, the peculiar stare will soon be noticed. If ptosis is present, the deformity of the drooping upper lid will soon become a source of annoyance, and the vision will, as I said before be soon interfered with, but only mechanically.

THE TYPICAL FEATURES AND SYMPTOMS are therefore:

1. COMPLETE IMMOBILITY OF BOTH EYES. This assertion has to be modified to the extent that the eyes will, in the beginning or even after the disease existed for some time have a slight motion sideways, and also downwards, if the patient makes a strong effort.

2. No impairment for distant vision, unless poor vision due to an error of refraction or to corneal opacities, existed before the attack; color perception remains good and the field of vision is not affected.

3. Slight inconvenience for work on small objects, reading or writing, due to loss of power of convergence; but the vision of each eye alone remains as it was before the attack and the power of accommodation does not suffer.

4. There is no visible change of the eyeballs themselves.

5. There is apt to be ptosis; but the drooping of the upper lid is, in some cases, so slight that it can hardly be noticed, whereas, in some, it is so marked that the patient cannot go about unless he forces his eyes open by contractions of the frontalis muscle.

6. The disease is progressive in so far as the ocular muscles become affected one after another, but after these are paralyzed, the disease remains at a standstill, and does not affect other muscles.

The HISTORY of my case is as follows: Mr. E., 30 years old, enjoying perfect health, found that he could not clean or cut his finger-nails without closing one eye and turning

his head. This happened in the fall of 1883, while he was in camp in the Adirondacks. He paid little attention to the fact because his distant vision remained as good as it ever was. In the spring of the following year, after returning to the city, his friends complained that he passed them in the street without seeing or recognizing them. It became likewise annoying to him that, when reading the newspaper, he had either to hold it very far from his eyes or to one side, and even then he had to move his head in order to read, for he could not take in a whole line at a glance. This made him think that there was something wrong with his eyes, and he came to me for glasses. On examining him, I found that he had a slight degree of hypermetropia, but that his vision was perfect in every respect, his color-perception and his field of vision were good, but when I came to examine his muscles with prisms, I found that he had monocular vision, and that there was no convergence of either eye. The left eye was quite fixed, but the right one moved slightly in a downward direction, and a very little inward. There is a slight ptosis of both eyes, a little more marked on the left side, but the patient has acquired the habit of keeping his eyes open by contraction of the forehead to such an extent, that the drooping of the lid is not apparent on superficial examination. There is likewise a very slight divergence of the left eye, which he uses for near vision, whereas the right eye is used for distant vision, although both eyes have equally good visual power. The pupils are moderately small, but act promptly if accommodating, for light and on convergence. Power of accommodation perfect; patient reads fine print in a region from three to twenty inches.

In all other respects the patient enjoys perfect health; has never had headaches, and only the hay fever drives him to the mountains. He has no business and spends most of his time in travelling about. About fifteen years ago, he had an attack of gonorrhœa, which was followed by some symptoms pointing to a specific infection, while there had been no outward initial lesion. For the last ten years I have seen him almost constantly, and he has had

no specific symptoms ; but in the spring of 1879, and likewise in 1880, he had an eruption on the back of both hands that lasted several days : it was very itchy, and disappeared after the use of little rhubarb and bicarbonate of soda. The family history of the patient is good, although on his mother's side there is a decided nervous tendency and scrofulous condition, dating from his great-grandfather, but they live, as a rule, to good old age. Patient is a high liver and fond of a good cigar, smoking as many as fifteen of them a day, is unmarried, and a man of excellent physique.

The treatment consisted in the use of the faradic current and the administration of iodide of potash ; this drug was given for nearly eighteen months, and at times in very large doses ; one hundred and fifty grains were given daily for several weeks. After this it was discontinued, and strychnine given instead, but with little or no result. The ptosis only has improved, and the action of both trochleares is much better than it was for some time. Patient attributes this to the systematic exercise that he gives his eyes daily.

In conclusion, I would state that, inasmuch as there has not been a single autopsy in plain, uncomplicated binocular ophthalmoplegia, the theories in regard to the initial lesion are simply hypothetical. Of the most recent researches, those of Mauthner point to a nuclear origin, whereas Bristowe and others consider it a functional affection ; but the probability that it depends upon changes of nutrition of the nuclei or perhaps of some centre of associate movements of the eye, is likewise probable, and when Strümpell emphasizes that the absence of diplopia is due, in all probability, to a diseased condition of ganglion cells presiding over the associate movements, why not go a step farther and say that the whole trouble is due to it ? The relation of the trouble to progressive bulbar paralysis and general atrophy is very doubtful to my mind, for the reason that the trouble is usually confined to the eye muscles, and that even these do not remain constantly paralyzed, but recover at times partly, or, as in one of Mauthner's cases, entirely. The probability that the corpora

quadrigemina might be the seat of the affection, is not a strong one, because in the post-mortem case of Warner, made by Bristowe, they were found to be normal on microscopic examination.

## ELECTRICAL CHARTS.

BY M. ALLEN STARR, M.D., PH.D..

PROF. OF DISEASES OF THE NERVOUS SYSTEM, NEW YORK POLYCLINIC.

THE graphic method of recording symptoms in disease has many advantages. It presents to the eye at a glance a series of facts which otherwise would require tedious description. It enables a comparison to be made with ease between varying or successive conditions. It portrays the course of an affection in a way which is easily grasped, either by the student, the attending physician, or the uninstructed patient himself. The universal adoption of the use of temperature charts attests the value of the method in watching the symptom of fever in all acute affections. The sphygmographic tracing is taken in many cases of cardiac and kidney disease, and enables one to measure the varying changes in heart action or arterial tension with accuracy. Any further application, therefore, of this method hardly needs urging. It will commend itself at once.

Some years ago, Erb drew up in graphic form the changes in electrical reaction occurring in various forms of paralysis. His charts, which are reproduced in the text-books both of nervous diseases and of electro-therapeutics, are familiar to all who employ electrical measurements in diagnosis. The object of this paper is to urge the employment of these charts more widely in the general treatment of cases of paralysis, both for the purpose of simplifying and making clearer the history of such cases, and, what is of greater importance, for the purpose of determining the prognosis. It is to this latter point that attention is called.

It is always important to be able to state with approxi-

mate accuracy the duration of a disease. It is absolutely necessary to determine in a case of paralysis whether recovery is possible and how soon. In some cases, the diagnosis of the nature and seat of the lesion enables one to give the desired information. In other cases, it will not. For example, in a cerebral hemiplegia one can judge, from the severity of the onset, the extent of the paralysis, the occurrence of contractures, and the rapidity of the reaction and re-establishment of function, as to the prospect of a cure and as to the duration of the loss of power. But in cases of peripheral paralysis, this is by no means true. For example, in lead-palsy, or in multiple neuritis of alcoholic or other origin, recovery will probably occur. But some cases remain for a long time stationary; others progress rapidly; and in all, the constant question asked is: "When shall I be well?" This question cannot be answered on general principles. One patient with lead-palsy may be told that he will probably be able to work again in three months, and at the end of two years he may still be in a stationary condition. Another may be told that he cannot recover for a year, and at the end of three months he is well. This is a rather mortifying position for the physician in either case.

It is especially in this class of cases that the graphic method of electrical measurements will be found of service. It is well known that in the majority of serious cases of peripheral nerve lesion, faradic contractility is abolished, and it is only to the interrupted galvanic current that muscles contract. The intensity of the current required to cause such contraction varies greatly in different cases, and at different stages in the course of a single case. Now, if in any case measurements of that intensity are made at equal intervals for some weeks, and the successive measurements are recorded graphically and joined by a line, an "electric curve" can be produced upon the chart. Such a curve must be either: 1st, horizontal, in which case the condition in the electric reaction of the muscle is stationary; or, 2d, it will be toward the normal point, in which case the condition is improving; or, 3d, it will be



away from the normal point, in which case the condition is getting worse. In the second case, in which improvement is in progress, it will not be difficult to determine quite closely the time of perfect recovery, since, after three or four measurements, the curve obtained can be projected to the normal point. It is usually the case that voluntary power is regained in a muscle some weeks before the electric reactions become normal. Hence it may be that a recovery of power can be promised quite definitely at a certain date, when the curve is obtained.

In all cases, the measurements must, of necessity, be recorded by the aid of an absolute galvanometer in milliamperes; for any conclusions drawn from a comparison of the number of cells of the battery employed is fallacious, since the strength of the cells is a varying quantity. Thus in one case it was ascertained that, when the battery used was newly filled, eight cells were sufficient to obtain a reaction in one of the paralyzed muscles, while, the day before, fourteen cells had been needed to produce the same result. Actual measurement by the galvanometer, however, demonstrated the intensity of current to have been the same on both days. The chart should, therefore, be made from accurate galvanic measurements.

If the faradic current is employed, a scale should be so attached to the coil as to indicate the strength of the current. The Dubois-Reymond standard coils are not to be obtained of our electric-instrument makers, and are expensive when imported. A simple means may be employed for such measurements if the secondary coil be made to slide over the primary coil, and the distance through which it is pushed be marked in millimetres. Where the secondary coil is wholly removed from the primary, and there is no induced current, 0 is marked, and from this point toward the primary coil the scale is laid down.<sup>1</sup>

The following chart will illustrate these statements:

The vertical divisions correspond to the various dates of measurements. The horizontal divisions in the upper

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<sup>1</sup> Waite & Bartlett have made such a coil for me.

scale indicate milliampères, the measure of intensity of the galvanic current. The double line indicates about the normal intensity of current needed to cause a contraction, though this has been found to vary between one and three milleampères.<sup>1</sup> The horizontal divisions in the lower scale indicate millimetres of overlapping of the two

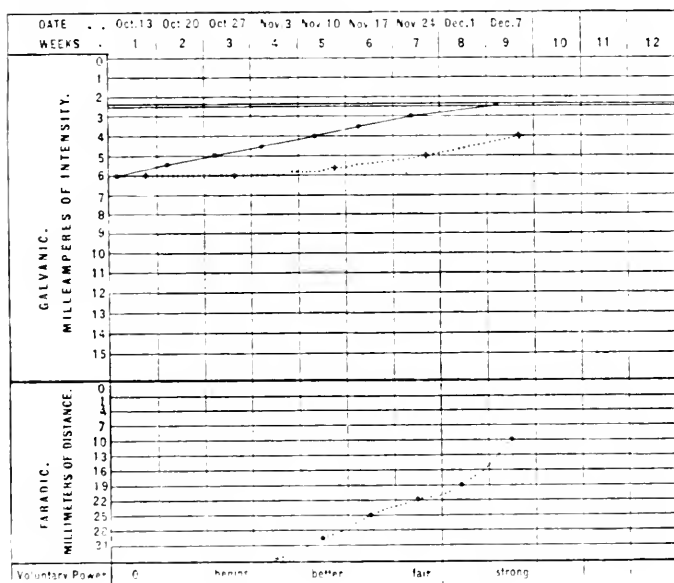
*Electric Chart.*

Name: C. E.

KCC -----

Muscle: Ext. Com. Digit. Sinister.

AnCC .....



faradic coils. In measuring the galvanic current, direct application of one pole to the muscle alone is considered, and not to the nerve, since it is this application which is made in treatment, and alone produces a response in severe cases of degeneration. A record of the reaction from the nerve may be added if desired. In the case which is figured on the chart (multiple neuritis), the measurements in the extensor communis digitorum sinister are given. The continuous line indicates the curve of negative closure contractions (KCC). The dotted line indicates the curve

<sup>1</sup> Mobius, "Allgemeine Diagnostik der Nervenkrankheiten," p. 144.

of positive closure contractions (AnCC). Both are below normal. At first both are equal, that is,  $KCC = AnCC$ , the same intensity of current producing contractions with positive and negative poles. Later, the  $KCC > AnCC$ , which is thus seen to approach normal. In the fourth week of treatment, faradic excitability returned, and gradually was produced by weaker currents. After four measurements in this case, it was possible to project the line and to state approximately the date of recovery. The case was seen many weeks after the onset of the disease, it had been stationary for some time, and a prognosis as well as a diagnosis was desired.

Those who are familiar with electrical measurements will notice that in this case there was a decrease in electric irritability, rather than the increase which sometimes occurs in degeneration. This I have found to be frequently the case in toxic paralysis. It is certainly true that the degeneration curve in lead-palsy differs widely from that in paralysis from pressure, though both are peripheral in nature. It is not at all unlikely that the use of such charts as that here presented will enable curves to be determined characteristic of various forms of degeneration. I have not used the charts long enough to establish conclusively various curves. It is partly the object of this paper to urge their immediate employment by those who are constantly applying electricity, with a view to determining such curves from a number of cases. The charts of Erb are diagrammatic, like the text-book curves of typhoid temperatures. It is not unlikely that a record of numerous cases by this method will afford interesting facts of importance, in diagnosis as well as in prognosis. Such charts have proved of great service in recording cases of fever. May they not be equally useful in cases of paralysis?

## NOTE ON THE SPECIAL LIABILITY TO LOSS OF NOUNS IN APHASIA.

BY MARY PUTNAM JACOBI, M.D.,

NEW YORK.

SOME months ago, it occurred to me that it would be interesting to ascertain in how many cases of aphasia the defect bore upon any particular part of speech or mode of speaking. For this purpose I examined the records of one hundred and sixteen cases, and found that, among them, in seventeen the patient had only lost the memory of noun substantives, or the faculty to employ these in voluntary speech. They were replaced by a periphrase, in language often quite fluent. Among the ninety-nine remaining cases, in only two was any other part of speech systematically affected. In one, the patient had lost the adjective, but she had also lost the noun. In the other, the patient had lost control over pronouns, some of which, however, were used, but improperly, and only employed the infinitive of verbs. The seventeen cases are as follows :

CASE I.—Broadbent describes a patient, aged 77 at the time of death, who was seen at intervals between 1878 and 1883. His infirmity dated from a slight and fugitive attack of right hemiplegia, predominating in the face, and accompanied by hemianæsthesia. There was at first a somewhat general disturbance of speech which finally became restricted to the loss of nouns. This defect persisted five years. During all this time the patient never uttered a noun but once or twice, and then inappropriately ; could say anything else, and employ long phrases, so that they did not contain a noun. When he wished for anything he would say, " Please give me the one."<sup>1</sup>

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<sup>1</sup> Med Clin. Trans., 1872.

CASE II. was another of Broadbent's, similar in all respects to the first, except that the patient could not read.<sup>1</sup>

CASE III. was also a patient of Broadbent's, a gas inspector, aged 59. The first complaint of this patient was that he found himself unable to read, and could not remember names of places, persons, or things. Pointed to legs and arms, and said he forgot the names of these. On another occasion, said that he could not recollect the name of this, taking hold of his coat. The doctor said "trousers." He said at first "yes," but then said "coat." Asking him afterward to name his finger, he muttered "coat, hat, boot," then was silent. I suggested thumb. He said, "yes, thumb," but afterwards "finger." This same patient was able to give a lucid description of an accident which had happened to him seven or eight years before.<sup>2</sup>

CASE IV. is less striking, because the entire faculty of speech was much more compromised: The patient had a few favorite routine expressions, as "Ca va bien; un petit mieux." He could not repeat the name of the objects shown to him, and made fruitless efforts to do so. If he were told the name, however, he would recognize it as correct; make a sign of affirmation, and observe, "oui, c'est ça." But he could not repeat the word himself.<sup>3</sup>

CASE V.—(Case of Dr. Allin, reported by Drs. Ball and Seguin.) The patient, after a third attack of cerebral accidents, recovered power of speech to a considerable extent, but had much difficulty with proper names and common names. Of a glass of milk he would say, "That is something to drink." Would have flashes of fluency on various subjects. With the progress of convalescence, the patient's vocabulary increased.

CASE VI.—Trousseau describes an eminent lawyer who had the habit of frequently forgetting the name of the thing about which he wished to speak. Addressing his wife, he would say, "Give me then my—my—*sacré matin*, my—you know very well." Then he would raise his hand to his head. "You want your hat?" "Yes, my hat." On another occasion, as he was going out, he rang the bell. "Give me my um—*sacré matin*!" "Your umbrella?" "Yes; my umbrella."<sup>4</sup>

CASE VII.—Bateman<sup>5</sup> quotes from Bergman (*Zeitschrift für Psych.*, 1849) the case of a man, who, after a fall, lost the memory of proper names and common substantives. He retained memory of verbs, and was able by means of periphrases to express his meaning.

<sup>1</sup> Med. Times and Gazette, 1885.

<sup>2</sup> Ibid.

<sup>3</sup> M. F. Balzer, *Gaz. Méd. de Paris*, 1884.

<sup>4</sup> Archives of Medicine, 1881, vol. v.

<sup>5</sup> Peter, *Gaz. Hebdom.*, 1864.

<sup>6</sup> Aphasia, 1878.

CASE VIII.—The same author also quotes from Graves (*Dublin Quarterly*, Feb., 1851) the case of a farmer, who, after an attack of hemiplegia, could no longer employ nouns in his speech, though he always remembered the initial letter.

CASE IX.—This was one, observed by Bateman himself, three years after accidents, which consisted exclusively in the sudden loss of speech. At the time of observation, the patient was able to talk, but not to use substantives except incidentally. Thus on being shown a purse, remarked: "I can't say the word; I know what it is; it is to put money in." Here it is noticeable that, although the noun which was required as the object of the proposition could not be remembered or pronounced, yet another noun, money, referred to incidentally, could be named.

CASE X.—Lasegue<sup>1</sup> describes a priest from Canada, aged 65, who could relate his own history fluently, but used no nouns, or only with the greatest difficulty.

CASE XI.—Lordat relates the case of the naturalist Broussonnet, who only retained the use of two nouns, soir (evening), which indicated the future; and juments (mares), by which he referred to a lady and her daughter. He replaced all other nouns, common or proper, by periphrases, or by a series of adjectives. Thus he called one friend, "He whom I love well;" and another, "The great, good, modest one."<sup>2</sup>

CASE XII.—At an Academie discussion in 1873, Bouillard mentioned a man, known to Cuvier, who had lost the memory of nouns, but was able, nevertheless, to compose phrases regularly and completely.<sup>3</sup>

CASE XIII.—Piorry quotes the case of an abbé who had lost the memory of nouns. He would say "give me my, that which one puts on the—" then point to his head, showing that he meant his hat, or else "give me that which is worn to clothe one's self."

CASE XIV.—Bernard quotes another case from Bateman, where the patient, instead of scissors, would say, "that with which one cuts," and for window, "that by which one sees," or "that where it makes light." In this second expression, as in another case already quoted, the patient used a noun incidentally (light), but could not do so with deliberate intention.

CASE XV.—A patient of Gairdner's called Monday, "the first working day," his aunt, "his nearest relative on the mother's side."<sup>4</sup>

<sup>1</sup> *Annales Méd. Psychol.*, 1877 (Soc., Feb. 26th).

<sup>2</sup> Quoted by Bernard, "De l'aphasie," 1885, p. 185.

<sup>3</sup> *Compt. rend. Acad. des Sciences*, t. lxxvii., 1873.

<sup>4</sup> *Arch. de Méd.*, 1866, 6e S., t. viii.

CASE XVI.—This is described by Dingley. Five weeks after a slight attack of hemiplegia, patient was obliged to use circumlocutory phrases to describe objects. Thus, whenever shown the picture of a camel, he said, "Egypt long way."

CASE XVII.—Lichtheim relates a case of word deafness, where the patient talked a good deal in a flowing manner, though with some tendency to repetition of the same phrases, but he always had the greatest difficulty in naming objects, and assisted himself by descriptive phrases. Thus, for wine he would say, "that is strong;" for water, "that is weak."<sup>1</sup>

From the foregoing list are excluded the much more numerous cases on record where the patient used the wrong nouns to express his meaning. For obvious reasons are also excluded cases where the entire vocabulary was extremely restricted.

To any one who first begins to examine the records of published cases, it might seem as if a much larger number could be collected of any given peculiarity. But all remarkable cases have done service many times, by being quoted over and over again by different authors, so that much care in verification is required in order to avoid repeating one case as several.

The peculiar form of aphasia under consideration has attracted much attention. Lasègue declared that the loss of the noun, "the substance of the discourse," was the most characteristic circumstance of aphasia.<sup>2</sup> Bouillaud called attention to this peculiarity in his communication to the Academy in 1873; Chevreul, following, offered an explanation of the fact. Falret, in 1866,<sup>3</sup> Bateman, in 1870 (quoting also an explanation by Osborne), Voisin in the "*Nouveau Dictionnaire*;" Bernard, in his monograph in 1884, all note that if any grammatical part of speech is systematically lacking to aphasics, it will invariably be the noun. Kussmaul,<sup>4</sup> I believe, makes a separate category of such partial aphasias, as do also Broadbent<sup>5</sup> and Licht-

<sup>1</sup> Brain, January, 1885.

<sup>2</sup> Loc. cit.

<sup>3</sup> "*Dictionn. Ency.*" Art. Aphasie, 1866.

<sup>4</sup> "*Die Störungen der Sprache in Greisen.*"

<sup>5</sup> Medical Times and Gazette, June, 1884, also Med. Clin. Trans., 1872.

heim.<sup>1</sup> "The loss of the noun," observed Ross, "is the most marked feature of sensory aphasia."<sup>2</sup>

The existence of this feature of language defect has sometimes seemed to conflict inexplicably with the common belief that children in learning to talk learn nouns first. It is then supposed that the noun must be that part of speech which becomes the most firmly "organized" in the brain, and should therefore be the last to disappear when the brain is injured. Yet the reverse is certainly observed.

The partial, or, as we may call it, the noun defect, is observed in amnesia (sensory aphasia). Case VI., from Trousseau's clinics, illustrates amnesia without aphemia; the patient forgot the names of objects, but when told this name, he recognized it as correct, and was able to pronounce it.

Case XIII. is precisely similar. In the other cases, it is not stated whether the patients were able to repeat the name which they were unable to remember. The impression is conveyed in the majority of the histories that this could not be done. When the spoken word was nevertheless understood, it is to be inferred that there was no serious defect on the motor side of the speech mechanism, and that the receptive, sensory side, was only incompletely injured. For in focal lesion of the auditory centre, spoken language sounds like gibberish to the patient. And where the power exists to repeat the word under the influence of the immediate stimulus from the auditory centre, this implies that the path between that and the co-ordinating centre of articulation is intact, and also that the latter centre is not seriously damaged.

Two general inferences must be drawn. 1st. That the lesion in these cases of partial defect is relatively slight. 2d. That it involves the paths connecting the auditory with the concept centre, or those which associate the latter with the motor co-ordinating centre. These conditions would be fulfilled by a moderate diffused lesion or pertur-

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<sup>1</sup> *Loc. cit.*

<sup>2</sup> "Handbook Dis. Nerv. Syst.," Philadelphia, 1885.



bation of the conducting tracts B M or A B in Lichtheim's schema.

"The lesion in amnesia," observes Lichtheim, "is not focal, but appears in more diffused morbid processes, or where cerebral circulation is deficient." The records of autopsies are not as useful as might at first be supposed, in solving the problems of this partial amnesia. To some of the most interesting recorded cases, no records of autopsies are appended. In the others, the lesions found belonged either to a period of disease from which the patient had partially recovered when he exhibited the partial defect, or to an exacerbation which preceded death, aggravated the symptoms, and determined the fatal issue.

Thus it is really more profitable at present to examine the question from the point of view of the mechanism of the naming process, considered in both its psychological and physiological aspect. Around the naming process have ranged some of the most celebrated controversies of philosophy. Whether the names of things, *i. e.*, nouns, were used first, as Dugald Stewart<sup>1</sup> asserts; or whether the first words were verbs, and indicated action, the theory of Adam Smith; whether common names were evolved from proper names, or the reverse; whether a class name represented a real existence apart from the individuals composing it; or whether it stood for a real concept, a conceivable notion of the mind; or whether it were strictly a sign for a collection of attributes, these being alone conceivable, such questions as these racked the brain of humanity centuries before the cerebral localization of speech was dreamed of. That the existence of a class name proved the existence of a real abstract being, an archetype upon which the individual members of the class were modelled, was the doctrine of the realists of antiquity and of the middle ages.

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<sup>1</sup> According to Dugald Stewart, the primitive men on seeing a wolf coming would cry, "wolf, wolf." According to Adam Smith, they would shriek, "he comes," and point to the beast in explanation (quoted by Max Müller, "Science of Language," p. 31.

But no one any longer supposes that the words man, or horse, or table corresponds to abstract but real beings, and this famous doctrine has no bearing upon the psychology of the naming process. It is otherwise with the second or conceptualist doctrine. This is constantly to be found cropping out, often unconsciously, from the most positivist descriptions of the mechanism of speech. In these, English physiologists, at least, usually assume the necessity of explaining, first, how a concrete or general idea or concept is formed from sense impressions, then how a name becomes attached to this idea. The mode of attachment is sometimes very oddly expressed. Thus, Ferrier is quoted by Hammond as saying: "The ideas of which words are the articulate symbols have no relation to that part of the brain where words are remembered, except by associating fibres."<sup>1</sup> We may justly ask what is meant by attaching an idea to any part of the brain. We might as well talk of connecting the time occupied by the run of a railroad train with the space it goes over. Broadbent,<sup>2</sup> in an analysis of the mechanism of speech, in many respects most admirable, observes: "The conception or idea of external objects is gradually formed by the fusion of the visual, tactual, and other impressions to which it gives rise. This idea is associated with an auditory impression which has been used to designate it." If for the term of "conception" we should substitute the other, "mental image," little would be lacking in Broadbent's description, at least from the standpoint of our present knowledge. Yet danger lurks in the term "mental image" also. The younger disciples of the purely materialistic school sometimes commit themselves to unintelligible absurdities by attempting too much precision in the history of "mental images."

Thus Mlle. Skwartzkoff, author of a good thesis on aphasia, and an article on word-blindness, describes the

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<sup>1</sup> West Riding Reports, 1874 (quoted by Hammond, "Dis. Nervous System," Eighth Ed., 1886, art. "Aphasia").

<sup>2</sup> "Med. Clin. Trans.," 1872, vol. lv.

evolution of the spoken word as follows: "Every object strikes several senses at once, and causes the development of as many sensitive images, whose totality constitutes the idea we have made for ourselves of this object. The impression forms in a first centre into a sensation, and this in a second cortical centre forms an image. The different sensitive images are *transmitted* towards the centre for the formation of words (foot of the third frontal convolution and surrounding parts), where the totality of these images takes its formula, its name. This name, by means of fibres of transmission, reaches the medulla, whence the nervous fibres animating the diverse parts of the apparatus of phonation project it outwards.<sup>1</sup> But what is a name that it can be thus transmitted on nerve fibres like a messenger on the string of a boy's kite?

In this connection it is well to remember the caution of Hughlings Jackson: "A method which is founded on classifications which are partly anatomical and physiological, and partly psychological, confuses the real issues." These mixed classifications lead to the use of such expressions as that an idea of a word produces an articulatory movement; whereas a psychical state, an "idea of a word" (or simply a "word") cannot *produce* an articulatory movement, a physical state. . . . We must keep these several sides of our subject apart, considering now the psychical side—speech—and at other times the anatomical basis of speech.<sup>2</sup>

Speaking, then, exclusively on this anatomical basis, we may say, with Broadbent, that impressions made by the object upon the various perceptive centres of the brain, fuse together, after converging upon some cell area intermediate to these centres, into a complex impression of this object. When the object has been named at the time it was perceived, an auditory impression is made simultaneously with the visual and tactual impressions, and this fuses together with the rest. Now it is possible to revive the mental image of the object by reviving any one of the

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<sup>1</sup> Mlle. Skwartzkoff, Archives de Neurologie, 1881, t. II.

<sup>2</sup> Brain, Oct., 1878. "On Affections of Speech," Hughlings Jackson.

original impressions, or even the memory of these. Among these means of revival, that of the auditory impression or name is so frequently made, and has so many conveniences, that it becomes the habitual sign of the rest; and the name is used to draw into the consciousness of the person speaking or of the person addressed, all the secondary or revived impressions of the sense attributes of the object.<sup>1</sup> "The word," observes Whitney, "is simply the survival of the fittest, among a variety of resources, (gestures, etc.) for effecting the same purpose, namely, the mental revival of the attributes of an object."<sup>2</sup> Thus, as Taine remarks, the association of a name with an object creates a *couple*, formed on the one hand by an auditory sign, on the other by the group of attributes with which the sign is associated. Of this couple, either member has the power of bringing the other into consciousness; and, the first extension of mental processes becomes possible when the sign may be substituted for the thing, and handled apart, like a mathematical symbol.<sup>3</sup>

In these descriptions, the word "impression" is used with an intentional vagueness, to cover the unknown molecular processes which take place in the cortical sensory centres, in the intermediate cell areas, of, as Broadbent suggests, the non-sensory, the superadded convolu-

<sup>1</sup> "Whatever performs the office (of directing our attention to particular elements in the perception) is virtually a sign; but it need not be a word: the process certainly takes place to a limited extent in the inferior animals; and even with human beings who have but a small vocabulary, many processes of thought take place habitually by other symbols than words. . . . In many of the familiar processes of thought, and especially in uncultivated minds, a visual image serves instead of a word." John Stuart Mill, "Examination of Sir William Hamilton," 1865, vol. ii., p. 73.

<sup>2</sup> Whitney, "Life and Growth of Languages," 1882. The author remarks that speech has the preference over gesture, even when it is less forcible and explicit, because it leaves the hands free.

<sup>3</sup> "On Intelligence." Am. transl., 1872, p. 6. "In the formation of couples, such that the first term of each suggests the second term; and in the aptitude of this first term to stand wholly or partially in place of the second, so as to acquire, either a definite set of its properties, or all those properties combined, we have, I think, the first germ of the higher operations which make up man's intelligence."

tions, and in the innumerable tracts of nerve fibres which associate these together. Of these processes, we can only frame to ourselves a schematic representation. While for some purposes the term "images," answers well enough in this schema:<sup>1</sup> for others it is misleading, and the conception of a molecular vibration answers much better. It certainly is much more in accord with such analogies for nerve action as we are almost compelled to draw from the physical phenomena, sound, light, and electricity.

The phenomena of musical combinations afford a guide at least for the schematic description of the name-evolution. The sound of the spoken name is certainly produced by air vibrations, which mediately impress the auditory nerve, and conceivably throw its molecules also into vibration. We may represent to ourselves these vibrations as continued to the cortical auditory centre, and there determining others, which, according to the special lines of intercellular fibres that are traversed, cause, what Broadbent has called the specialized grouping of cells. These are not, of course, displaced in the nerve mass, but brought diversely into relation with each other, in the same way as battery cells scattered through a laboratory may be diversely grouped according to the wires included, at any given moment, in the circuit. As far as our present data carry us, such a specialized vibration in the auditory centre would suffice to bring the sound of the spoken word into consciousness. The "fusion" of this vibration with others analogous, coming from the visual and tactual centres is, as we must conceive it, analogous to the fusions of small groups of musical vibrations into larger groups, producing more complex sounds. This complex vibration, occurring in the so-called concept centre of Lichtheim, the superadded convolutions of Broadbent, does not "*produce an idea*;" it is itself the physical side or substratum of one phenomenon of which the conscious impression, idea, image, or concept, is the psychic aspect. The concept again, is not, as Sir William Hamilton declared, something conceivable by the understanding,

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<sup>1</sup> It is constantly used by Meynert.

though not by the imagination; <sup>1</sup> but so far as it means anything, it is the *equivalent* of the mental image, or the psychic aspect of the complex vibration. This mental image differs from each sensory image by the very fact of its complexity, and also by its probable formation in non-sensory portions of the brain. It is these anatomical localities, and not the ideas, which are connected with the sensory centres by association fibres. Finally, the auditory impression or vibration does not become a name in the auditory centre; but only after it has become an integral part of the complex, fused vibration, whose psychic aspect is the idea or mental image. Hence a name in an unknown language is gibberish. The same consideration shows that the name is not affixed to the idea of an object after that has been separately elaborated. It is possible, it is true, to perceive an object whose name is unknown to the percipient. But, if the latter wish to communicate any impressions of this object to another person, he must make use of some sign to indicate it, and the sign, though but an indicative gesture, is already the essence of the word, and is simply replaceable by a verbal sign when that shall have been suggested. In the absence of communication, actual or potential, there is no language.

Although a concrete name be the sign for a real mental image, composed of the remembered attributes of the object named, a general name is not. It is here that the modern philosophic doctrine of nominalism becomes identified with the modern physiological doctrines of speech and thought. The philosopher may declare that there is no abstract conception in the mind, the physiologist that there is no material image in the brain, no matter how refined and etherealized. It is impossible to have an abstract conception of a triangle that shall be free from any peculiarities of some individual triangle, as a scalene or isosceles, etc. But it *is* possible to abstract the property of three angledness from a class of figures, of which each individual possesses this, though possessing other proper-

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<sup>1</sup> "Lectures on Metaphysics."

ties besides. It is this property or attribute that is recalled to the mind, and which the mind is capable of contemplating apart by means of the special verbal sign—triangledness—attached to it. "Thus," observes Hamilton, "a sign is necessary to give stability to our intellectual progress, to establish each step in our advance as a new starting-point for our advance to another beyond. A country may be overrun by an armed host, but it is only conquered by the establishment of fortresses. Words are the fortresses of thought."<sup>1</sup>

The internal mental image becomes realized in speech through further propagation of these (supposed) cerebral vibrations toward the point where they can determine such grouping of nerve cells as can secondarily regroup cells in the ganglionic centre immediately presiding over organs of phonation, that is, towards the corpus striatum. All recent testimony tends to localize this point of convergence at the foot of the third left frontal convolution. The considerations which precede, suffice to show, however, the absurdity of regarding this convolution as the "seat of the faculty of language." Broca himself only claimed that lesion of this convolution was followed by "loss of the memory of the means of co-ordination that are employed to articulate words."<sup>2</sup>

The far greater extension given to-day to the total cerebral mechanisms employed in speech render superfluous the criticisms upon Broca's doctrine which are based on the discovery of lesions of parts of the brain other than his convolution, and which have been found to co-exist with some form of aphasia.<sup>3</sup>

I have not found any record of cases which show a loss of power to articulate names, when it was clear that these

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<sup>1</sup> Quoted by Mill, loc. cit., p. 68.

<sup>2</sup> P. Broca, Bull. Soc. Anatom., 1883, t. viii. (quoted by Bernard, loc. cit., p. 175.

<sup>3</sup> Thus Hammond, in the latest edition of his treatise, reproduces a table published by Seguin in 1868 (*Quart. Journ. Psychol. Med.*, Jan., 1868), containing eighteen autopsies called in favor of Broca's theory, and thirty-four against. This merely refers to the cases with and without lesion of the third frontal convolution.

could be spontaneously recalled by the patient, when, at the same time, other parts of speech could be articulated.

When an object, or a class of attributes constituting an abstract conception, can be recalled to mind, but its name cannot, it is evident that the visual and tactual perceptions of the objects have persisted, while the auditory impression, or else its point of fusion with the rest, have been effaced. Chevreul says that this has happened because less attention has been paid to the name than to the sense attributes of the object. Ross, following Hughlings-Jackson, says that names disappear first in the dissolution of speech, thus in the mildest cases, because they are less well organized knowledge than that of simple relations.<sup>1</sup> I think there is another reason, which may be rendered clear by considering the primitive development of speech. It is highly improbable that this began in the use of either nouns or verbs, but rather in conglomerates, shorter or longer, which constituted an entire proposition. Children, in learning to speak, use words at first with precisely this complex significance, and it is a matter of accident whether the word employed be a noun, verb, adjective, or even a preposition. I knew a little boy extremely intelligent, but who, at the age of two years, could only say five words, yet contrived to express himself wonderfully well by gestures. But one of his few verbal signs was "hard-a-lee," an expression that he had learned while sailing, and which he habitually used either to refer to a sailboat, to urge a wish to go sailing, to announce his possession of a boat to a new-comer, etc. The verbal conglomerate was not learned first because it was simple or easy, for it was neither; but it belonged to the circumstance that had made the most forcible impression on the baby's mind.

According to Renan, many primitive languages abound in conglomerate expressions. The Greenlander treats an entire phrase like a single word, and conjugates this word like a simple verb. Among the majority of the North Ameri-

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<sup>1</sup> Loc. cit.



can Indians, continues the same author, the composition and agglutination of words is pushed to an almost incredible extent. Each phrase of these languages is only a verb, in which all the other parts of the discourse are inserted.<sup>1</sup> In the successive experience of both the individual and the race in the acquisition of speech, the order would seem to have been as follows: 1st. There are the sensory impressions made by the object. 2d. A proposition arises in some one's mind, to be communicated about this object to another person by means of a verbal sign, more or less extensive in significance, but probably always at first unique. 3d. There is a gradual breaking up of this conglomerate sign into words occupying special relations to each other.

Whitney observes that the establishment of a clear distinction between the noun and the verb especially marks the genius of the Indo-European languages, and it is not nearly so well marked in others.<sup>2</sup> Thus, although the hypothesis be provisionally useful for the purpose of analysis, it is probably not really correct to say that the process of naming ever consists in fusing a verbal sign merely with the sensory impressions of a single object. The conglomerate verbal sign was evolved from original interjectional sounds, under the pressure of a strong desire of communication with a fellow-being. For this very reason, the sign must always have implied a proposition concerning the object referred to. So long as the primitive man simply recognized the wolf, and took his own precautions for defense, there was no language. Language began when men began to concert together for defense against a common enemy. The very least that could then be said was, "There is the wolf," or "the wolf comes," complete propositions involving a subject and a predicate, but both probably expressed together by a single conglomerate sign. This sign represented the fusion of an auditory impression, not only with the group

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<sup>1</sup> "De l'origine du Langage," Sixth Edition, 1874, p. 156.

<sup>2</sup> "Life and Growth of Language."

of visual impressions which made up the general mental image of the wolf, but with the visual impressions of events in which the wolf took part. At the present day, though the original conglomerate be broken up into separate words, the phrase still retains its unity in thought. If from lesion of the associating fibres through which diverse impressions may be fused, this unity is weakened, and the phrase threatened with dissolution, the part which first tends to disappear is that which is most easily replaceable by visual image. This is certainly the part of the phrase or conglomerate sign which indicates the object itself. The speaker can point to it when in sight, can describe it by periphrases when it is out of sight; but such replacement is possible for nothing else in the proposition. As long, therefore, as speech is possible at all, it will express by verbal signs those parts of the proposition which cannot be expressed in any other way, while the name which can be diversely suggested is forgotten as a simple sign.

Temporary forgetfulness of a name is, as is well known, not at all uncommon among quite healthy people. Any one, by observing himself closely in these cases, may recognize that the difficulty of recalling the name seems to be directly proportioned to the clearness of the visual image of the object. As an example: I found myself the other day telling a person to go down on the piazza, and stammering over the word "piazza," while I was at the same time picturing to myself the locality with unusual distinctness.

The patients who recall the names of objects that are incidentally imbedded in the phrase describing an object whose name they cannot recall, illustrate the theory here, advanced. When such a one says, upon seeing a purse, "I know what it is, but cannot name it; it is to put money in," the noun, "money," is merely part of an adjective phrase which might be expressed "it is money-containing."

The name recalls the properties of money so faintly that the visual image of this object cannot triumph over the verbal sign and obliterate it. But the object in view, the

object of the entire proposition, excites a visual impression so much more powerful than the auditory sign belonging to it in the verbal conglomerate—the phrase—that this sign is obliterated. It is not, of course, that the visual impressions or memories are absolutely increased in strength; they become relatively stronger simply because the mechanism for the revival or for the association of all verbal impressions is damaged, and these, therefore, are weaker.

It seems to me that this theory is much better grounded than that which attempts to distinguish between the words which “are better organized in the brain,” and those which are less so. No auditory sounds, however highly specialized, are words, until they are understood as the signs of things or of the relations of things. And no words are, in themselves, any fixed part of speech, but only exist as words in the relation they occupy to the mental grouping of the moment.

It is this relation which first disappears in sensory aphasia, while enough of the mechanism for recording verbal auditory impressions remains to enable the patient to recognize a name pronounced before him. The association of this verbal sign with the visual impressions of an object may be so much damaged that revival of the one in consciousness will not recall the other. The psychological difficulty depends on physical injury to the anatomical tracts which connect the visual and auditory centres.

In the conglomerate mental image framed of the object and of a proposition concerning it, there will persist the reminiscence of the sense impressions of the object and of the auditory signs used for enunciating the proposition. These signs have never been connected with any particular visual impression, but only with a series of relations whose memory is registered or organized in the concept, supra-sensory centre. In the milder forms of sensory aphasia, the paths between these intellectual centres and the auditory centre on the one hand, and the motor centre on the other, are presumably intact; no dislocation takes place between the auditory signs and the series of relations

to which they correspond. The name of the object is, however, entirely dislocated from its habitual associations; the impulse or vibration which passes from the visual centre goes directly to the concept centre, without fusion with any impulse coming from the auditory centre. The final mental conglomerate of the proposition, therefore, which is to be expressed, consists partly of reminiscences of sense impressions, partly of revived verbal signs, instead of being composed entirely of verbal signs, as is normal. The verbal signs which remain in the conglomerate are repeated by the articulatory mechanisms which receive the appropriate stimulus to functional cell grouping. The visual reminiscences of the object cannot be expressed by these mechanisms any more than waves of sound could be reproduced by the retina, or waves of light by the auditory nerve. This substantive portion of the conglomerate proposition can only be expressed by gestures or by visual signs. Such signs must have served the purpose of expression before any auditory signs had become specialized into speech. They serve such purpose again when auditory signs have become disassociated with objects on account of lesion of the anatomical paths through which visual and auditory impressions may fuse together.

It has been suggested to me by a friend who listened to the exposition of the foregoing theory, that, in accordance with it, abstract nouns, as "love," "patriotism" "virtue," should be retained by the aphasics in question, because they are associated with no definite visual image, but with series of relations. It would be interesting to test this suggestion.

## Clinical Cases.

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### RECOVERIES FROM INSANITY IN CASES ACCOMPANIED BY HÆMATOMA AURIS.

BY CARLOS F. MACDONALD, M.D.,

MEDICAL SUPERINTENDENT, NEW YORK STATE ASYLUM FOR INSANE CRIMINALS.

Cases of insanity complicated by *hematoma auris*, or "insane ear," have so rarely terminated in complete recovery that the development of this complication has come to be regarded as sufficient to warrant an unfavorable prognosis in every case. In fact, authorities upon insanity, almost without exception, lay it down as a rule that a recovery is not to be expected in such cases; and it is possible that the acceptance of this belief may have led physicians to relax in their efforts to promote recovery, as is wont to be the case in diseases that are known to be incurable. Hence the importance of recording every favorable exception.

In the *Am. Journal of Insanity* for July, 1874, Dr. E. H. Van Deusen, then Superintendent of the Michigan State Lunatic Asylum, reported three cases of complete recovery from insanity associated with marked and unmistakable *hematoma auris*.

The first of these cases occurred in the practice of Dr. William Teats, of the Coton Hill Institution, England, who reported it in the *British Medical Journal*, 1873.<sup>1</sup> The subject was a woman, aged 33 years, admitted to Coton Hill Institution in January, 1870, suffering from acute mania, first attack of about three months' duration, and characterized by marked suicidal, homicidal, destructive, and violent tendencies, and improper language. The case was regarded as hopeless. This condition continued until August, 1872, when the excitement gradually subsided, and by the middle of November she was considered perfectly sane.

The second case, reported in the same journal by Dr. Needham, of the Bootham Asylum, at York, was that of a young man, admitted to the Bootham Asylum in October, 1861, with acute mania of one week's duration. At the end of twelve months no improve-

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<sup>1</sup> Dr. Van Deusen believes this to be the first case ever recorded.

ment had occurred, and the patient seemed to be rapidly drifting into dementia; there had been hæmatoma of both ears, with the characteristic effusion, absorption, and subsequent disfigurement. Suddenly there was a change for the better; the excitement abated, habits and mental condition began to improve, and at the end of sixteen months the patient was discharged fully recovered; and up to the time of making the report, upwards of ten years, he had remained perfectly well.

The third case was observed by Dr. Van Deusen himself. A robust young farmer, aged 21 years, was admitted to the Michigan Asylum in June, 1870. He was suffering from acute mania of about three weeks' duration; the attack was attributed to "partial sunstroke and business perplexities," and was characterized by extreme violence, marked bodily agitation, and destructive tendencies. Five weeks after admission, well marked hæmatoma occurred in both ears, passing through the usual stages, and terminating in the characteristic deformity. "In October, there was an abatement of the excitement, convalescence was established, and he was discharged recovered, February 15th, 1871. His health has continued good, as has been ascertained by occasional correspondence, and he has been actively engaged in business since his return home."

The following interesting case occurred in my own practice: Geo. C., *alias* P., aged 18 years, convict, was admitted to the N. Y. State Asylum for Insane Criminals, April 16th, 1880, suffering from acute mania of a somewhat violent type and of several weeks' duration. He recovered, and was discharged from the asylum November 11th, 1880; was readmitted on June 22d, 1881, at which time he was in a markedly maniacal state, continuing so during a greater part of the year. He was incoherent in speech, noisy, violent and filthy, smearing himself with excrement, denuding his person, and openly masturbating; also attempted self-mutilation. In August, 1881, commencing hæmatoma of both ears was observed. These developed rapidly, were apparently painless, and fluctuated indistinctly—the right being the larger. The swelling, in each, was confined to the concha, giving a very peculiar appearance. Absorption was rapid, and at the end of a month the tumors had nearly disappeared, leaving the usual induration and thickening.

At the same time the mental disturbance had gradually subsided, and convalescence was fairly established. The patient was returned to Auburn prison, recovered, September 30th, 1882, where he remained entirely free from mental disturbance, as I ascertained through occasional visits to him, until the expiration of his term of sentence to penal servitude, March 22d, 1884, when he was released.

These four cases comprise, I believe, all that have thus far been reported, although Dr. Clouston, in his admirable work on mental diseases, referring to *hematoma auris*, says: "I have seen only

three cases recover out of eighty cases who had *hæmatoma auris*.”<sup>1</sup> And again, the same author, in describing a case of adolescent insanity with *hæmatoma auris* followed by recovery, makes the following statement: “This was one of only about six patients that I have seen where recovery took place after a *hæmatoma* had formed, or even been threatened in any degree.”<sup>2</sup> Just how Dr. Clouston would explain this apparent discrepancy, the writer is unaware; most likely it is a typographical error.

The foregoing cases are here presented in the hope that others, having similar cases, may be induced to report them, and that, in this way, sufficient evidence may be obtained to warrant a more hopeful prognosis, and, consequently, a more assiduous application of curative measures in cases of insanity complicated by *hæmatoma auris*.

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<sup>1</sup> “Clinical Lectures on Mental Diseases,” p. 265.

<sup>2</sup> Op. cit., p. 380.

## REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILADELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF W. H. WALLACE, M.D., PHYSICIAN-  
IN-CHIEF, AND CHARLES K. MILLS, M.D., CONSULTING PHYSICIAN.

CASE VIII.—*Mania—Swelling and Extravasation of Blood into the  
Right Cheek, analogous to Hæmatoma Auris.*

Reported by Harriet Brooke, M.D., Assistant Physician.

The patient was a middle-aged woman who has occasional attacks of mania, with lucid intervals, sometimes lasting years. Recently, a marked change was noticed in her manner and she soon became very maniacal. A little before the mania became marked, a large swelling appeared on her right cheek, immediately over the malar bone, and there was a large extravasation of blood beneath the skin covering the tumor. The patient had received no violence from the attendants, and she had not done herself any violence. The tumor appeared suddenly, *i. e.*, through the night. An old attendant, who had seen her during previous attacks, said that her face had always swelled at such times. As the mental trouble subsided, the swelling also disappeared, leaving the skin for a time shrivelled and with a slightly pemphigoid eruption, which in time also disappeared.

This case is of considerable interest. We do not recall any reports of similar cases. The assistant physician who makes the report is very certain that it was not due to traumatism. The fact that the swelling occurred at the beginning of the period of mania also lends some color to the idea that it was connected with the disease as a vaso-motor phenomenon, rather than the result of injury.



## THROMBOSIS OF AN ARTERY IN THE TEGMENTUM OF THE CRUS CEREBRI.

By M. ALLEN STARR, M.D.,

PROF. OF NERVOUS DISEASES, NEW YORK POLYCLINIC.

Henry B——, age 40, applied for examination at the New York Polyclinic February 27th, 1886, complaining of numbness in the right side of his body and of double vision. He denied syphilis, but admitted having had gonorrhœa twice, and having suffered from severe sore throat and nocturnal headache. Has never had any eruption. No scars on genitals. It is, therefore, uncertain whether he has had syphilis.

He was fairly well until five months ago, when, on waking one morning, he noticed that he saw everything double, and had a peculiar sensation of numbness in his entire right side. This condition has been permanent.

Examination shows a well-nourished man, able to walk naturally. There is slight ptosis of the left eye, and a marked sinking downward of the eyeball when at rest. The pupil is slightly dilated, but reacts to light and during accommodation. Voluntary motion of the eyeball is good, except upward, the superior rectus being weak. When his eyes are at rest, he sees double, the new object appearing just above the old one. From the consequent misinterpretation of the position of objects, he lifts his feet higher than necessary in walking or in going up-stairs. When he turns both eyes to the right or to the left, the double vision persists, but the two images maintain the same relative position. There is no apparent deviation to either side, and no secondary lateral deviation can be noticed when one eye is covered. Exam. by Dr. Webster: R.  $\frac{2}{3} \frac{0}{0}$ , L.  $\frac{2}{3} \frac{1}{0}$ . Hm  $\frac{1}{3} \frac{0}{0}$  in both eyes. No insufficiency at 20', nor at 1'. Ophthalmoscopic examination negative. The condition of the eye is one of paresis of the levator palpebræ and superior rectus muscles; all other muscles of the left eye and all muscles of the right eye being normal.

He complains of a constant feeling of numbness and tingling in the right half of the body; face, arm, body, and leg being equally involved. Examination shows a slight degree of tactile anæsthesia in the entire right half of the body, excepting a small area back of the ear and on the neck, where sensation is normal. There is also a difference between the perception of temperature sensations in the two halves, heat and cold being perceived less intensely on the right side. The sense of pain is also somewhat impaired on the entire right side. These sensations are not delayed in transmission. Muscular sense is normal. There is no incoordination, all motions being accurately and promptly performed. The reflexes are normal and equal on both sides. There is no loss of power. There are no other symptoms. Vertigo and headache are absent. Hearing normal. The examination indicates a lesion

in the course of the sensory tracts of touch, temperature, and pain from the right half of the body, not sufficient in extent to destroy completely that tract, but only to impair its action.

Since the sensory and visual symptoms began simultaneously, it is probable that they are due to one cause. The fact that but two of the branches of the left third nerve were involved points to a lesion in the nucleus of the nerve rather than to a lesion in the nerve trunk. It is interesting to find that both Völkers and Kahler locate the nuclei of the levator palpebræ and superior rectus muscles side by side. The nuclei of the third nerve lie in the following order: 1st, the centre for accommodation; 2d, the centre for the pupil; 3d, the centre for the rectus internus; 4th, the centre for the rectus superior; 5th, the centre for the levator palpebræ; 6th, the centre for the rectus inferior; 7th, the centre for the obliquus inferior. In this case, the fourth and fifth centres were affected. But just at the side of the gray matter lining the aqueduct of Sylvius, in which these centres lie, the *formatio reticularis* of the *crus cerebri* is situated. In the *formatio reticularis*, the tracts conveying touch, temperature, and pain pass from the opposite side of the body toward the internal capsule of the cerebrum.<sup>1</sup> A lesion lying in the *formatio reticularis* on the left side of the tegmentum, if small in size, might at once interfere with the transmission of sensations from the right side of the body, and affect the nuclei of the left third nerve. The *lemniscus*, lying outside of the *formatio reticularis*, would not be affected if the lesion were a small one, and hence no ataxia or inco-ordination would be present. A lesion, therefore, in the *formatio reticularis* of the tegmentum of the left *crus cerebri* would explain perfectly the symptoms present in this case.

As to the nature of the lesion, its sudden onset and the stationary character of the symptoms indicated that tumor and hemorrhage were equally improbable. If a small thrombus had formed in one of the twigs of the posterior central artery which supplies the *crus cerebri*, it would have led to a small area of softening, the symptoms of which would have been sudden in onset and stationary in character. An embolus would produce the same effect, but the patient's freedom from cardiac disease threw doubt on the hypothesis of embolism. The diagnosis of thrombosis was strengthened by the fact that syphilis was suspected, an endarteritis being the usual cause of thrombosis.

The case is interesting, as it demonstrates the possibility of local diagnosis from the peculiar combination of symptoms present; and also as it is rare to find an affection of but two of the nuclei of origin of the third nerve. The failure of the patient to return a second time prevents any statement regarding the termination of the case. No case in any way resembling this one has been found after long and careful search in the neurological journals of the past ten years.

<sup>1</sup> "The Sensory Tract in the Central Nervous System," by M. A. Starr, *Jour. Ment. and Nerv. Dis.*, July, 1884.

## Periscope.

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### THERAPEUTICS OF NERVOUS SYSTEM.

#### The Effects of Hyoscine Hydrobromate and of Hyoscyamine in Insanity.

Dr. Henry M. Wetherill, Jr., of the Pennsylvania Hospital for the Insane, in a paper appended to the report of the Lunacy Committee of the State Board of Public Charities, Pennsylvania, 1885, presents, in tabular form, some observations of the action of hydrobromate of hyoscine and hyoscyamine as used in the hospital with which he is connected.

There are three tables in all, comprising, respectively, 10, 7, and 12 cases, total 29. Table 1 shows the effects of hyoscine hydrobromate (Merck's), administered per os at 9 P.M., in cases of insomnia. All the cases in the group, mostly chronic lunatics, were habitually insomniac, and all the usual means to produce sleep had proved unsatisfactory. They were so under observation that it was positively known whether they did or did not sleep. The dose ranged from  $\frac{1}{100}$  to  $\frac{1}{240}$  gr., and the period of observation was three weeks. The results as to sleep were almost uniformly good, the period of sleep varying from five hours to all night.

Table 2 shows the effects of hyoscyamine (Merck's), pure, crystallized, white, etc., in insomnia. This group comprises six cases of chronic, and one of subacute insanity. The drug was administered by the mouth, and at 9 P.M., as in the cases of table 1, the dose varying from  $\frac{1}{12}$  to  $\frac{1}{60}$  gr. The results as to hypnotic effect were not nearly as satisfactory as in the first group, where hyoscine was used, the periods of sleep varying from none at all to all night, with a preponderance in favor of the shorter. Dr. W. suggests that an instructive comparison may be drawn from these tables as to the relative hypnotic activity of the two drugs.

Table 3 is more elaborate. It shows the effects of hyoscine hydrobromate (Merck's) in a group of cases of chronic insanity, habitually in a state of aggressive violence, destructiveness, and great motor activity, and accustomed to the usual modes of medication. Horizontal lines show the record of the patient for the day, and three observations during that time are recorded for each

patient, the conditions of pulse, respiration, skin, pupils, etc., being carefully observed. The results as shown in this table were highly satisfactory.

"It might be in place here to say that we have made a thorough trial of hyoscyamine (Merck's), pure, etc., in the wards, in this same class of cases, with gratifying results, in hypodermic doses ranging from gr.  $\frac{1}{80}$  to gr.  $\frac{1}{12}$ . It is our experience, however, that the dose has to be rapidly increased, and that it is a weaker and much less satisfactory remedy than the hyoscyne hydrobromate. Taking, then, the latter as the subject of the following remarks, it would appear to be a useful agent in the treatment of insomnia and of motor excitement. Our experience has extended to many more cases than those included in the tables, and it appears probable that it will be found applicable to all the forms of mental disorder which embrace as symptoms sleeplessness and restlessness. In cases of acute mania, the continuous effect of small doses, given, preferably, by the mouth, will be found useful in subduing morbid activity. In the agitated form of melancholia, in doses varying in accordance with the urgency of the symptoms and with the strength of the patient, it has been found to act very satisfactorily. The writer is, at this time, engaged upon the study of its action in a group of cases of acute insanity, and the results will be tabulated and offered for examination.

"Thus far, it is believed, no fatality has resulted from the administration of hyoscyne, but it is an agent that should be used cautiously. We have found that, in a few instances, it has not been well borne—very moderate doses having been followed by extreme relaxation, anxiety, præcordial oppression, spasmodic pain referred to the cardiac area, and by weak, irregular, and intermittent action of the heart. Such extreme effects from moderate doses are exceptional. Its conservative action is, doubtless, the most valuable of its effects. While it is very desirable to subdue or modify the noise and confusion of an excited ward of chronic patients, occasioned by the influence of its most aggressive elements upon the others, it is highly important, in a more hopeful class of cases, to economize the vital forces and to prevent excessive tissue waste by controlling insomnia and motor excitement. This being accomplished, the case is buoyed over its most critical period with the minimum amount of exhaustion, the convalescence is more rapid and satisfactory, and the period of residence in a hospital is abridged. It is evident that hyoscyne, intelligently used, will fulfil these conditions. It is no exaggeration to state that the excited wards have entirely changed in character for the better since the introduction of this remedy, and its beneficial results in acute cases have been very encouraging. We hope that this agent will receive fair trial at the hands of the general practitioner of medicine. It is not uncommon to hear of obstinate cases of insomnia whose disorder resists the most careful and varied treatment. This condition continuing, undoubtedly predisposes to attacks of mental disorder. In this condition hyoscyne might prove useful. The

general practitioner is frequently brought into contact with cases of insanity in the very commencement of the attack, and is called upon to combat the insomnia and morbid restlessness over which the remedies usually prescribed exert but little influence. It is possible that very serious consequences might be averted if, in connection with generous feeding, sleep and relaxation of motor energy could be induced. The nervous perturbation present in cases of morphia habit, of intemperance, or mania-a-potu, and of delirium occurring in the course of a certain physical maladies, would suggest the possible usefulness of this drug. The writer recalls a case of delirium closely simulating mania-a-potu, from rapid abstraction of morphia, in which the hypodermic use of pure hyoscyamine, in doses of one-sixtieth of a grain, produced the happiest effect. We have found the use of hyoscine hydrobromate, in doses of one one hundred and twentieth of a grain, very useful in the insomnia following alcoholism.

"So far as our investigations have proceeded, we have found hyoscine to be a valuable therapeutic agent, and it continues to be in daily use in the medical service of this hospital. It seems probable that, after thorough trial and investigation, its range of applicability will be extended and it will recommend itself to the profession as one of the most valuable discoveries of modern chemistry."

The effects of hyoscyamine and hyoscine hydrobromate as observed by Dr. Wetherill are substantially in accord with those noted by the medical officers of the Auburn asylum, where the drugs are not a little used for the relief of insomnia, motor excitement, etc. Another effect of these remedies, however, has occasionally been noticed at Auburn, mention of which the reporter does not recall having seen, namely, visual hallucinations, supposed to be due to influence of the drug upon the sight centres. In the reporter's experience also, in a certain proportion of cases, these drugs have utterly failed to produce a hypnotic effect—the hyoscine in doses as high as  $\frac{1}{30}$  gr., and the hyoscyamine,  $\frac{1}{10}$  to  $\frac{1}{8}$  gr.

CARLOS F. MACDONALD.

### Cure of an Hysterical Contracture by Hypnotic Suggestion (*Archives de Neurologie*, September, 1886).

In this article, Voisin describes a case of hysterical contracture cured by him by hypnotic suggestion. The patient was an ignorant peasant, and there was no possibility of deception. She was 40 years old, and for two years had had nervous crises with retrosternal sensation of *globus*, constriction of the œsophagus, retention of urine, and other manifest hysterical symptoms. After one of these attacks, she became paralyzed in her right arm six months before she was seen by Voisin. Several days after the attack, a fresh one followed, which produced the contracture of the right hand, and this had persisted. Patient was intelligent, memory good, speech clear; had had no troubles before these at-

tacks. The right arm that had the contracture gave every appearance of a cerebral paralysis of long standing. It was absolutely inert, and incapable of the slightest movement; the forearm was drawn into a state of semi-flexion, and all the fingers were forcibly flexed. The nails were buried in the palms, and these were hollowed with ulcerations, which exhaled a fetid odor; the phalangeal articulations were tumefied and painful. All attempts at extension caused sharp pains, and seemed to increase the contraction. Sensibility of all kinds preserved; there was no muscular atrophy. The other members, trunk, and face, presented nothing abnormal, neither paralysis nor anæsthesia. Excepting the absolute lack of power in the right arm, the patient was well and appeared in good condition. From these facts, Voisin concluded that he was dealing with an hysterical contracture, and decided to try hypnotic suggestion. Patient was thrown into a hypnotic sleep by fixation of the eyes after twenty minutes; the sleep was complete with analgesia and complete flaccidity of all the members. He then commanded her in a loud voice to extend the little finger of the right hand; this she did with some difficulty and with a manifestation of pain. Then she was ordered to open the ring finger. She did this, too, but the difficulty and the pain were still greater, and the suggestion had to be very energetic. The other two fingers were easily opened. The extension was then almost complete, although evidently interfered with by the swelling of the joints, but she could move her five fingers with rapidly increasing facility, and every trace of contracture had disappeared. The nails were exceedingly long and foul, the palms ulcerated and bloody by reason of prolonged pressure and maceration of the epidermis. The arm and forearm were still immovable. She was ordered to move them, being assured that she could do so; she succeeded, at first with pain, but soon the right arm moved as freely as the left. In April, four months after the cure, Voisin learned that since his hypnotization she had used both hands freely in her household duties and in working in the fields. The retention of urine, which he did not have time to attend to, still continued (he had seen her while travelling in a distant part of the country). He thinks that this retention would have disappeared if he had made the proper suggestion.

WILLIAM NOYES.

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### Castration in Nervous Disease.

The *American Journal of the Medical Sciences* for October, 1886, contains a symposium, in which Sir T. Spencer Wells, Alfred Hegar, and Robert Battey are the feasters. The matter of discussion is castration in mental and nervous diseases. Sir Spencer opens for the opposition with a most interesting account of the operation, but ends in what sounds like a thundering condemnation of the operation. In certain forms of uterine disease, he regards the operation as advisable, but for the cure of nervous dis-

ease he opposes the operation as unjustifiable, excepting in rare and exceptional cases. In his denunciation of the wholesale and indiscriminate custom of spaying women, which has been gradually spreading of late, Sir Spencer Wells will probably carry with him the sympathy of all broad-minded and educated surgeons.

His conclusions are summed up as follows :

The operation of oöphorectomy, or the removal of normal ovaries, is one which may be advised in some cases of uterine fibroids and in uncontrollable uterine hemorrhages.

That it is to be resorted to in certain malformations of the genital organs, deformities of the pelvis, and accidental obstructions of the vagina.

That the right to use it is only limited in cases of ovarian dysmenorrhœa or neuralgia, and only when they have resisted all treatment and life and reason are endangered.

That in nearly all cases of nervous excitement and madness it is inadmissible.

That it should never be done without the consent of a sane patient, to whom its consequences have been explained.

That the excision of morbid ovaries and appendages should be distinguished from oöphorectomy, and ought not to be done without the authority of consultation, as in most other cases of abdominal section.

That in nymphomania and mental diseases it is, to say the least, unjustifiable.

It will be noticed that the advisability of the operation for epilepsy, in which the best results have been obtained, is not passed upon.

Hegar understands "by the term 'castration' the removal of normal or degenerative ovaries, not, however, including those which have developed into large tumors." The general principles underlying the operation and the general pathology of the diseased conditions of the generative organs are then discussed with much diffuseness and equal dryness. General principles, rather than specific data, are dwelt upon. The final opinion seems to be that "castration is indicated in a psychosis evoked or maintained by pathological alteration of the sexual organs, and in a neurosis originating from the same source, as soon as this imperils life or hinders all occupation and all enjoyment of life." Other milder methods are to be tried first, etc., etc., "but castration is absolutely no universal remedy for any neurosis originating from a genital organ disorder, or kept up by the same." The operation will be of use when a degenerated and dislocated ovary represents the irritative focus, etc., etc. As the data upon which these opinions are founded are not given, the value of the opinions themselves cannot be well estimated.

Batley is more specific. He opens with the remark : "Within my knowledge, it has not been the practice of American surgeons to attempt the cure of mental and nervous disorders by the removal of healthy ovaries or of healthy tubes. The ovaries removed,

and the tubes as well, have presented visible signs of disease—signs which are evident to the naked eye and palpable to the sense of touch. For the misconception upon this point still existing my own ignorance of both the histology and pathology of the ovaries is largely responsible, in that during the early history of the operation I removed ovaries which I erroneously supposed to be healthy, and gave to the operation the unfortunate and now obsolete name of 'normal ovariectomy.'

"The operations now under consideration have been done for the relief of mental and nervous disorders, which may be divided into three classes, namely: oöphoro-mania (hystero-mania), oöphoro-epilepsy (hystero-epilepsy), and oöphoralgia." Battey uses the term "oöphoro-mania," etc., because he thinks that these disorders are dependent upon irritation proceeding from the ovaries, and not from the uterus. The time required for the disappearance of the nervous disorders, after removal of the ovaries, has been quite variable. In general, epileptiform seizures have ceased at once. Some of the cases have required bromide for a time, while others have needed nothing. His cases of mania have all been quite chronic and the improvement slow. In oöphoralgia, in a few instances, the cure has been immediate and permanent. In the majority it has been slow and gradual, and in others nothing has been gained for even two years after the operation.

In the cases which have had two or more years to test them, the results are tabulated as follows :

	Cured.	Improved.	Not Improved.	Total.
Oöphoro-mania, . . . .	1	4	2	7
Oöphoro-epilepsy, . . .	9	0	0	9
Oöphoralgia, . . . .	13	3	4	20
	<hr/>	<hr/>	<hr/>	<hr/>
	23	7	6	36

The removal or non-removal of one or both tubes has not affected the final results.

The duration of the cases of mania had been from three to fourteen years; of the epileptic cases, the majority were of one to three years' standing, and one extended to six years. The cases of oöphoralgia varied from three to twelve years in duration, but few of them less than five years.

The cases of oöphoro-epilepsy have given the most satisfactory results.

Dr. Battey concludes with a number of communications, with reports of cases from Wm. H. Byford, M. D. Mann, R. S. Sutton, W. T. Howard, H. P. C. Wilson, B. F. Baer, Wm. Goodell, T. A. Emmet, and T. Gaillard Thomas. The inference left to be drawn from these communications would seem to be that while the operation is liable to abuse, in properly selected cases it is, as Prof. Mann says, "not only justifiable but urgently called for, but that these cases are very few."

MORTON PRINCE.



## Book Reviews and Bibliographical Notes.

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**A Manual of Diseases of the Nervous System.** By W. R. Gowers, M.D., F.R.C.P. Vol. I., Diseases of the Spinal Cord and Nerves. Philadelphia: P. Blakiston Son & Co., 1886.

The prevailing interest among scientific physicians in the study of nervous diseases is proven by the large number of new treatises which have appeared within the past five years. And a comparison of the volume under consideration with any similar book issued five years ago will demonstrate the many changes of view, advances in knowledge, and modifications in treatment which the work of this period has made. Of all the manuals published in English up to the present time, this is the best; and it may be said to equal, in all respects, any work published in German. Gowers has long been known as one of the foremost authorities on nervous diseases in England. His works have been characterized by a clearness of conception and definiteness of statement which make their perusal pleasant as well as profitable. It is evident that he has a knowledge of the subjects upon which he writes, derived not only from wide reading, but from careful observation and from pathological investigation. It should be the effort of medical authors to bring clinical facts and post-mortem findings into harmony, and in this effort the author is pre-eminent. It is also the duty of authors to weigh carefully, from wide experience, suggestions regarding treatment, and here again Gowers shows a proper conservatism.

The special features of this volume are the concise and complete statement of anatomical facts necessarily preceding any consideration of nervous diseases, the admirable analysis of nervous symptoms in general, and the exhaustive discussion of the various diseases of the nerves and spinal cord.

The fact that Gowers' small treatise on localization of diseases of the spinal cord has passed through three editions within five years is evidence of his success in handling the difficult subject of nervous anatomy. In the sections in this book devoted to the anatomy and physiology of the nerves and spinal cord, a very satisfactory summary of our knowledge of these organs is presented. All the important facts are included, and there is a degree of detailed statement, including most recent views, both of the anatomy and physiology, hardly to be looked for in a general text-

book. And the clear conception of the facts is greatly aided by the large number of figures accompanying the text. The cerebro-spinal tracts are traced with much clearness; for example, the motor tract, which he considers in its entire course from the cortex of the brain to the muscles as composed of two segments, an upper and a lower. "Each consists of a ganglion cell above, a nerve fibre, and the terminal ramification of the latter. The upper "cerebro-spinal" segment consists of the cortical ganglion cell, and the pyramidal fibre which proceeds from the cell passes through the brain and cord, and ends by dividing in the spongy substance of the anterior cornu. The lower "spino-muscular" segment consists of the spinal ganglion cell, and the fibre proceeding from this passing through the anterior root and nerve-trunk to the muscles where it divides and ramifies on the muscular fibre. The elements of the two segments do not probably correspond in number. Each cortical spinal element is connected with many spinal muscular segments. It will be found that this conception of the motor path conduces to clearer ideas of many phenomena of diseases" (p. 116). It is a matter for congratulation that this manner of tracing functional tracts through the brain and cord has received the approval of the English school of physicians, and it is to be hoped that it will soon be adopted by their anatomists; for, to the adoption of this method long ago in Germany are due many of the modern advances in cerebral physiology. In tracing the sensory tracts in the cord, the well-known view of Gowers, based upon two cases in his practice, of ascending degeneration in the antero-lateral tract, is urged again, viz.: that in the lateral part of the cord, near the periphery, lies a tract which conveys sensations of pain. The slender basis of this assertion is, however, frankly admitted, although the more general view, that sensations of pain and temperature are conveyed upward in the gray matter, a view based on numerous cases of syringo-myelitis, is not emphasized. Wherever in the consideration of the physiology of the cord disputed points are met, there is observed a care in presenting various views, and an originality in proposing solutions of the difficulty, which contrast markedly with the positive assertions found in some other works of the English school.

In the discussion of nervous symptoms, there is an admirable summary of the action of the various muscles, and the consequences of their paralysis; also of the effects of paralysis of the various nerves in producing deformity. The facts and illustrations are largely derived from Duchenne's work, "*Physiologie des Mouvements*," but have never before been presented to English readers in such a complete manner. After a careful study of these pages (22 to 37), no one need make a mistake in the diagnosis of a case of peripheral paralysis. The diagnosis of nervous diseases, by the aid of electricity, is concisely stated, and some original diagrams, illustrating the use of electrical examination in prognosis, are especially interesting. It seems likely that electrical charts of a nervous case are to become as important as temperature charts in

fevers, both for diagnosis and for prognosis. A somewhat more detailed statement of vaso-motor and trophic symptoms might have been given, but the essentials are to be found under the discussion of individual diseases.

It is impossible in a limited review to mention the special features of the work in the sections upon individual diseases of the nerves and cord. We may notice, however, the chapter on multiple neuritis, which contains all the facts at present known regarding this newly-discovered affection; the chapter on locomotor ataxia, which includes a full discussion of the theory of the disease; and the chapter on ataxic paraplegia, under which title Gowers describes combined sclerosis of the cord.

In all these chapters, as well as in those upon other lesions of the cord, the description of the pathology of the disease is very exhaustive, and is accompanied by drawings of cases under the observation of the author. The work contains, therefore, important additions to the previously known facts regarding the diseases of the cord. In the chapter on chronic spinal muscular atrophy, several diseases hitherto considered distinct are grouped together, viz., progressive muscular atrophy, amyotrophic lateral sclerosis, and chronic polio-myelitis anterior. While pathological facts certainly point to a close relationship between these diseases, the clinical picture of each is usually so distinct at the outset that they deserve special consideration; and the number of cases of progressive muscular atrophy which go on to bulbar paralysis is so much greater than that which go on to amyotrophic lateral sclerosis that it is somewhat misleading to group the latter two together, and separate the former. The subject of arthritic muscular atrophy, to which little attention has been given in previous text-books, is clearly presented. The chapter on tumors of the cord is less satisfactory than others, and seems to have been condensed from lack of space.

In regard to the sections upon the treatment of various nervous diseases, it may be said that they are entirely honest. So many absurd claims of cures by the application of electricity in nervous diseases have been advanced that a wholesome reaction against the indiscriminate use of this by no means harmless agent has at last taken place. The presentation of the legitimate claims of electricity, as well as a frank statement of its uselessness in certain cases, is an admirable feature of this book. For example, in the acute atrophic paralysis of children, Gowers says: "Electricity has been strongly advocated and largely used in the treatment of this disease, and there is reason to believe that it is useful, although its influence has been much exaggerated. In no sense is it a curative agent, and there is no evidence that its application to the spine is capable of increasing the degree or accelerating the course of the recovery of the nerve-elements. Nor is it easy to obtain evidence of its influence over the muscles. If the wasting is rapid, this progresses in spite of daily and sedulous applications. Faradization is useless, since the muscles cannot respond to it; but to

the voltaic current they act readily, and their daily stimulation, by its means, tends to maintain their contractility. "This is, it is true, of no avail if no recovery takes place in the spinal cord, but in most cases some recovery in the cord does occur, and when, after months, the new elements regain the power of conducting the voluntary impulse, the muscles are in a better condition to respond to it if they have been regularly galvanized than if they have been left alone." Such a clear statement of fact sweeps away the fraudulent claims of cures in infantile paralysis, so often urged in the past, and will commend itself to every one who has used electricity with care in these cases. In discussing its application in myelitis and locomotor ataxia, he is equally frank in affirming that it has little influence in affecting the pathological processes. In the treatment of peripheral lesions, the use of electricity is warmly advocated, and clear directions are given as to the method of its application. It might have been well to have been more specific in regard to the strength of the constant current used, the necessity of proper galvanometric measurements, and the duration of the applications. Those who have De Watteville's admirable little work on electricity can, however, supplement what is lacking here.

While the printing of the book and its general appearance are attractive, it is to be regretted that the many figures, which are of great importance to the pathologist, are so poorly executed. Whether this applies to the English edition, as well as to the American reprint, we are unable to state. But wood-engraving has attained such a point of excellence in this country that there is no excuse for neglecting to provide for a proper production of cuts in a work of such importance.

If the second volume, which will treat diseases of the brain, reaches the standard of excellence attained by the first volume, there will be no necessity for the publication of another work on nervous diseases for some time to come. M. A. S.

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**A Brief Synopsis of the Various Points Involved in the Coarse Examination of the Brain and Spinal Cord.** By FRANCIS X. DERGUM, M.D., Pathologist to State Hospital for Insane at Norristown; Instructor in Nervous Diseases, University of Pennsylvania. **First Annual Report of Pathology Department.** By FRANCIS X. DERGUM, M.D., and IDA V. REEL, M.D., Pathologists. Reprinted from the Sixth Annual Report of State Hospital for the Insane, S. E. District of Pennsylvania, at Norristown, Pennsylvania, 1885.

These two brief monographs, from the laboratory of the State Hospital for the Insane at Norristown, Pennsylvania, should be cordially welcomed by every one who has the cause of scientific psychiatry at heart. The abundant material for pathological research which is furnished by State insane hospitals has, in large part, been neglected in Pennsylvania as in other commonwealths. The appointment of pathologists, both resident and visiting, for

the Norristown Hospital was a promise of good for the future; and both Drs. Dercum and Reel are deserving of great credit for the manner in which they have responded to the opportunities afforded them.

In the synopsis of *Coarse Examinations*, the points are presented in a terse, systematic manner. They indicate that the author of the pamphlet is a practical observer and worker. The autopsies of cases are as large a number, we are safe in saying, as have been given to the profession during the entire existence of some of our older State institutions for the insane. Not a few of the reports are unduly brief and somewhat imperfect; but in these cases special circumstances doubtless prevented fuller investigation. In a large number of the cases, brief but sufficient notes of microscopical examinations are appended to the accounts of the megascopic lesions. Clinical histories are not presented in connection with the cases, which is much to be regretted. Some of the autopsies reported are of especial value, as, for instance, Case No. XXXVII., a female, aged 45 to 50 years, who suffered from dementia interspersed with periods of excitement. In this case, a condition which is spoken of as *multiple angioma of the brain with subsequent calcification* was found. It is to be hoped and expected that the hospital which has thus so auspiciously begun its pathological work will continue it with augmenting ardor and interest.

M.

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**Elektro-diagnostik und Electro-therapie.** Von DR. E. REMAK. Reprint from *Eulenburg's Real-Encyclopädie* (Urban & Schwarzenberg, Vienna and Leipzig, 1886).

The reprint before us includes the two articles in Eulenburg's well-known Encyclopædia, on Electro-diagnosis and Electro-therapeutics. In ninety-four closely printed pages, the author has managed to array all the facts and most of the theories now accepted in medical electricity. Electro-physics are incidentally discussed, in addition to the subjects which the title indicates; this reprint constitutes, therefore, a fairly complete treatise on medical electricity, in many respects more complete than any text-book on medical electricity with which the reviewer is acquainted. Certainly no text-book can be said to give such a mass of information as this little pamphlet does. The references to the past and current literature are very full indeed. The most recent advances have been noted with great care, and so we find reference to Jolly's recent investigation into resistance, Erb's studies of the electrical conditions of the muscles in Thomsen's disease, Gessler's researches on the terminal motor-plates in their relation to the reaction of degeneration, Müller's electro-therapeutical suggestions, and Engel-skjõns fanciful electro-therapeutical laws, which are severely denounced. All these recent investigations and many more are discussed. The author's own, and the older Remak's investigations receive due, though not excessive, attention.

We have no mind to point out minute details of this little brochure. In passing, we wish, however, to note the statement on p. 36, that the reversal of the normal formula is not an indispensable factor in proving a R. D. In this country, undue stress has been laid upon this one phenomenon to the exclusion of others of equal importance. Unless the promised third edition of De Watteville's book should soon appear, the neurological student could do no better than to refer in case of need to these excellent articles, and we repeat that no text-book is likely to give such full references as are here furnished. Those thoroughly acquainted with the subject of medical electricity will appreciate the masterly manner in which Dr. Remak has performed a difficult task. B. S.

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### Editorial Notes and Miscellany.

The next number of this Journal will contain a full Periscope and the proceedings of late meetings of the New York and Philadelphia Neurological Societies. In deference to the writers of original articles, these abstracts and reports have been laid over for next month's issue. Present indications are, that if all departments of the JOURNAL are to flourish equally, we may be compelled to exceed the original limit of 64 pages for each number.

THE  
Journal  
OF  
Nervous and Mental Disease.

Original Articles.

THOMSEN'S DISEASE (MYOTONIA CON-  
GENITA.)

BY DR. GEO. W. JACOBY,

PHYSICIAN TO THE CLASS OF NERVOUS DISEASES OF THE GERMAN DISPENSARY OF THE CITY  
OF NEW YORK.

THE history of this peculiar affection has been so lately and so thoroughly given by Erb<sup>1</sup> in his monograph upon this subject, that it would be superfluous to repeat the same here. Erb has examined all cases reported as cases of Thomsen's disease, and has carefully sifted them. He divides them into three classes; the first containing only purely typical cases, the second those which are more or less dubious, those which do not agree in every important point, but which at the same time show a great similarity to pure cases, and the third those which, although showing the principal symptoms, the "myotonic disorder of the muscles," still, on account of their complication with other symptoms, cannot be considered as true cases of Thomsen's disease, but are probably cases of central affection.

It will not be amiss to quote the following short definition of the affection from Erb. "I understand by the typi-

<sup>1</sup> Erb. W., "Die Thomsensche Krankheit (Myotonia Congenita)." Leipzig, 1886.

cal form of Thomsen's disease those forms of disease which generally, under the influence of an hereditary or family noxiousness, already in very early youth show that disorder of movements which later becomes more pronounced. The occurrence after a period of rest, and during movements, of tension and stiffness of the muscles up to complete inhibition of movement, gradual dissolution of this tension by means of continued movement, until entire relief occurs. Prolongation of the tonic contractions, produced by energetic action of the will, and inability to voluntarily relax the muscles quickly. In connection with this a remarkably strong, hypertrophic development of the voluntary muscles, in direct contrast to their relatively small power. The muscles also showing certain characteristic changes in their mechanical and electrical excitability, particularly the prolongation of artificially produced contractions; all other functions being perfectly normal, the nervous system especially not showing any other disorder."

Among the purely typical cases Erb classes those occurring in Thomsen's own family (twenty-two or twenty-three; two of which, those of Dr. Thomsen himself and one of his sons, only having been described, and even these without any objective examination), cases described by Leyden (one), Seeligmuller (two), Bernhardt (three), Peters (one), Strümpell (one), Petrone (one), Westphal (two), Ballet and Marie (one), Weichmann (two), Rieder (one), Knud Pontoppidan (four), Romain Vigouroux (one), Pitres and Dallidet (one), Eulenburg and Melchert (four), and Fischer (one), making in all, with Erb's cases, twenty-eight. Under the heading of doubtful cases he classes eleven.

The only case in the English language referred to is that of Engel and this case is classed as dubious.

Engel's case<sup>1</sup> bears in many respects a certain resemblance to a true case of Thomsen's disease, but it also

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<sup>1</sup> H. Engel. "A Case of Thomsen's disease. A form of Paresis of Motion accompanied by muscular hypertrophy." *Phila. Med. Times*, Sept. 8th, 1883.



differs in so many important particulars that it must be classed under some other heading. The points which show its diversity are the following :

The affection occurred suddenly in a youth 17 years of age in consequence of fright, caused by lightning striking a tree near the place where he was standing. The electro-muscular contractility is increased in the legs alone. Of this it is said that the response is quicker and the contraction stronger than in the other muscles, but not a word is said of prolongation or tonicity of the contractions. The muscular sense is impaired. There was present a mild foot clonus. A gradually increasing resistance was present to passive motion.

In the morning after getting up he has no difficulty whatever. The arms are not at all affected, and finally the description of the patient's manner of walking and falling differs entirely from that in Thomsen's disease. Engel says, "When told, after he had been standing erect for a long time, to walk, he lifted his left leg with great difficulty, and when the front part of the foot touched the floor the thigh was bent inwardly at the hip and the leg bent backward at the knee. Then the other leg underwent the same peculiar motion, and while this was going on, the upper part of the body swayed to and fro, a symptom which became worse the moment the other foot touched the ground ; then, with steadily increasing difficulty, the same process was repeated at the next step, the swaying of the body increasing still more, and at the commencement of the third step, the left foot gave way, gliding under the right leg, while the opposite took place with the right foot, so that the patient suddenly fell forward," etc., and further on, "almost the same phenomenon happened after he had been sitting awhile, only that then the upper part of the body swayed to and fro, from the moment he arose, and before he made the first attempt at locomotion," etc.

In various American journals, references are found to "Hamilton's cases of Thomsen's disease." The only article to which these references can allude is that published

by Dr. Allen McLane Hamilton, of this city, in the *New York Medical Record*, p. 85, 1886, and entitled "A Consideration of the Thomsen Symptom Complex with Reference to a new form of Paralysis Agitans." It is clear from a perusal of this article that Dr. Hamilton does not regard these cases as genuine cases of Thomsen's disease, but as cases of central trouble presenting certain symptoms of the former affection. Dr. Hamilton, in a letter to me, says that this interpretation of his article is in accordance with his views.

The case which I desire to describe was referred to me on Oct. 28th, 1886, by Dr. A. C. Bucklin, of this city, whom he consulted in reference to his eyes, and was shown at the meeting of the New York Neurological Society, Nov. 2d, 1886.

George H. Glenn, from Cape Vincent, Jefferson County, N. Y.,  
æt. 24, painter.

The family history is unsatisfactory, and nothing important from an hereditary point of view is discoverable. He does not remember his parents, who both died when he was a child. He never had any brothers or sisters, and his only living relative is an aunt who is perfectly healthy. He thinks that his maternal grandfather had some nervous trouble. He is unable to assign any cause for his infirmity. The affection has existed as long as he can remember, and he is unable to recollect any time when he was perfectly well. He remembers distinctly that he could not participate in the out-door games of his comrades on account of stiffness of his muscles. He could not run like other boys and was debarred from joining in athletic sports. He attempted to learn to play upon the organ, but was unable to make any progress on account of the clumsiness of his hands. What he particularly noticed was that all of his muscles were stiff, and that the execution of every movement was impaired. This was particularly noticeable after periods of prolonged rest. The first few movements always decidedly increased this stiffness, but then it gradually diminished and finally entirely disappeared, so that a perfectly free use of the muscles set in. This stiffness always reappeared after a pause or rest. All the voluntary muscles were affected, those of the upper as well as the lower extremities. Also the muscles of his face are involved. When he attempts to whistle he finds difficulty in doing so. In mastication, the muscles become stiff and rigid, and he is affected, as it were, with a temporary trismus. This stiffness was also noticeable in closing his eyes. Only such muscles or groups of muscles as were at the time employed would become thus affected, all the inactive mus-

cles remained perfectly lax. This tonic contraction of the muscles was, as far as he remembers, never accompanied by any pain. He thinks that he was decidedly worse when a boy than at present. If in running he stubbed his toe against any hard protruding object, all the muscles of his body would stiffen up like a board and he would fall, being unable to rise for several seconds. This does not occur now. At about the age of fourteen, he began to learn his trade, and he attributes his improvement to being continually at work, for, he says, "moving around and working limbers me up," and, "if I did not work now, but just kept quiet, I would be as bad as formerly." There were always periods of time during which he was better than at others. Mental influence did not have any effect upon his bodily condition. He says that he does not grow worse when thinking of it, or when excited. I desire, however, here to remark that when I presented him before the members of the New York Neurological Society, he was decidedly worse than I had ever seen him before, and I attributed this circumstance to the mental excitement caused by this presentation, although he himself would not acknowledge the correctness of my explanation. He also noticed that his muscles were large in comparison to other boys', and was astonished and chagrined to find that he had so little force in them. He also noticed a difficulty in bringing his eyes back to a certain point, and frequently after looking at some object situated far to one side for a short space of time, and then trying to look at something in front of him, he found that for a few seconds he would either not be able to see at all, or he would see double, but that by a shake of the head he would "bring his eyes around all right." About two years ago, after having been considerably troubled in this way and upon regarding himself in the mirror, he found that his right eye was "turned in." In every other way he was perfectly healthy.

*Status præsens.*—Patient is a person of medium height and strong bony structure; he has not a thick panniculus adiposus. His skin is of normal appearance. The shape of his body is normal, except in the exceedingly strong development of nearly all of the muscles. This at once attracts attention, being noticeable even with his clothes on. When undressed he looks decidedly athletic. Particularly strongly developed are the muscles of the leg and thigh, those of the gluteal region, and those of the arm and forearm. The muscles of the shoulders and neck are also extraordinarily prominent. The entire appearance, excepting that of size, is, when undressed, herculean. The face, however, does not show any of this hypertrophy. The following measurements will give an idea as to the size of the limbs: Right calf, 15 inches; left, 15½ inches. Right thigh, 7 inches above patella, 23 inches. Left thigh at the same place, 23 inches. Forearm above, right, 11 inches; left, 10½ inches. Arms, middle (over the biceps), extended, right, 12 inches; left 11½ inches; flexed, right, 14 inches; left, 13½ inches. Thorax over xiphoid process, 35 inches. Thorax over nipples, 36½ inches.

The heart, lungs, and abdominal organs are normal in every particular. Temperature and respiration also normal. Respiration when voluntarily increased does not show any difficulty; but expiration after a forced inspiration is occasionally difficult and protracted. The urine is normal. Joints freely movable. The nervous system was found normal in every way. Psychical condition, intelligence, and memory appeared to me to be normal. The patient himself says that his memory is poor, but I was not able to satisfy myself upon this point. Hearing good. Eyes and speech present certain anomalies to which I will again recur.

The sensibility of the skin over the entire body is normal. No vasomotor changes. No affections of the bladder. Cremaster, abdominal, and plantar reflexes present. No foot clonus. Tendon reflexes can be obtained from the patellar and triceps tendons, and occasionally also from the tendo Achilles. The patellar tendon reflex presents certain curious features which must be specially mentioned. Sometimes, especially if the reflex has not been tested for a long period of time, the first blow produces a decidedly exaggerated contraction of the muscle. This reflex contraction then diminishes with each successive blow, becomes normal, less than normal, and at times entirely disappears. The exaggeration is not always demonstrable, but the gradual diminution from the normal to total disappearance can generally be easily obtained; even to this, however, there are exceptions. All in all, the condition of this as well as of the other tendon reflexes was found to vary at various times.

The *muscular system* is the part in which the most noticeable changes are observed. Besides the great hypertrophy, the most apparent symptom is a certain stiffness and slowness of all movements which are executed after a period of rest. The movements which demonstrate this most forcibly are those of flexion and extension, and the patient himself notices the increasing stiffness as it is coming on, but before it is fully developed. If then the same movements through which the stiffness has been produced are repeatedly executed, this stiffness is gradually lost, and the patient can execute the previously inhibited movement without any trouble. If the patient is asked to execute any forcible movement such as forced flexion of the hand, of the arm, or of the leg, a tonic contraction of the exerted muscles takes place which lasts for some time after all influence of the will has disappeared. This tonic contraction is so great that for the time being all antagonistic movements are rendered impossible. This same phenomenon is observed after forcible closure of the eyelids, when he is unable to open them until after the lapse of many seconds. The power of the muscles stands in marked contrast to their enormous development, for it is easily shown to be decidedly diminished. Examination with a dynamometer is interesting, for it not only demonstrates the reduction of muscular force, but also shows the characteristic inability to relax the muscles which have been employed in the

pressure, as shown by the slowness and awkwardness with which the dynamometer is released from the grasp.

This highly interesting and pathognomonic symptom is present in all of the voluntary muscles of the body. The muscles of the lips are sometimes affected when the patient attempts to whistle or to forcibly pronounce a labial as P or B. In eating, that is to say in swallowing, it is occasionally difficult to get the bolus down; "it sticks in the throat." The tongue is also sometimes affected when he first begins to talk; for the last two statements I have taken the patient's word, I myself have not observed them. The movements of the eyes are also sometimes affected. Upon the right eye there is strabismus; this and other interesting features connected with the eyes are explained in the following extracts of a long letter from Dr. C. A. Bucklin, of this city, by whom the patient was kindly sent me. Dr. Bucklin, after carefully going over a variety of conditions which are likely to produce convergent strabismus, for this patient is affected with the convergent form, says:

"I am certain that no case of strabismus ever developed in a person who could see distinctly and with comfort at all distances with both eyes. This patient could not see at all distances with both eyes, because he was prevented from co-ordinating his visual axes by the tonic spasms of his internal recti muscles. For many years he would see double at every sudden movement that he made with his eyes; by closing his eyes for an instant, then upon opening them and giving his head a sudden rotating movement in both directions, he would find a point where he could fix with both eyes and see distinctly. As years went on he discovered that it was more and more difficult to make the diplopia disappear, owing to the fact of his internal recti muscles being so much stronger than the other ocular muscles and their also being more frequently affected by this spasm.

"These muscles finally began to shorten, so that he could only occasionally relax them and dispel the double vision. One day he accidentally discovered that he could voluntarily ignore the image of the deviating eye; from this moment he made only occasional attempts to see with both eyes, owing to the extreme difficulty of doing so. When an object is brought sufficiently near to make it convenient for him to see with both eyes, he fixes binocularly, and is perfectly conscious of seeing with both eyes. When the fixed object is suddenly removed two feet away he gives up fixing with both eyes and promptly declares that the deviating eye is no longer of any use to him. It is an interesting fact that he is conscious of the change from monocular to binocular vision.

"The vision of each eye is  $\frac{2}{3}$  on a bright day; it is not improved by any form of lenses. If he is asked to fix an object at the extreme right or the extreme left, some seconds elapse before he can fix upon an object in any other position. Both pupils react to light rather sluggishly. The deviating eye has a slightly larger pupil than the other eye, which reacts to light still more sluggishly. In a dark room he sees more distinctly with the deviating

eye than with the other one. Under these circumstances he frequently uses the deviating eye instead of the fixing one.

"The fundus is normal.

"The mobility of both eyes inward is abnormally great, their mobility in all other directions being normal. It is thus seen that Thomsen's disease may simply be another cause to disturb the natural relations existing between fixation and accommodation."

Fibrillary tremor in the muscles is sometimes present, but, besides this, I have twice noticed, once in the deltoid and once in the biceps, a lifting up and vermiform movement of entire groups of muscular fibres. There is also present a certain amount of muscular restlessness, so that the patient finds it difficult to sit quiet very long without moving about in his chair. Compression of the nerves or arteries does not produce any muscular contractions. The hypertrophied muscles are elastic and full to the touch, but there is nothing to be found which could be looked upon as an induration; the impression which they make upon the observer's hand is entirely different from the muscles in pseudo-hypertrophic paralysis. The joints are freely movable and passive movement does not produce any contractions of the muscles.

*Mechanical excitability.*—The nerves themselves do not show any abnormal mechanical excitability; the muscles, however, show a marked exaltation in this respect. I have not been able, as Erb has in his cases, to produce any contractions by simple pressure with the fingers or by rolling the muscle between the hands, but the application of an Esmarch bandage (applied for the excision of a piece of the quadriceps femoris), produced violent tonic contractions of the muscles of the entire leg. If, however, the muscle is given a sharp blow, as with a percussion hammer, this mechanical hyper-excitability becomes very manifest. When such a blow is given, a deep groove is formed in the muscle, running for some distance from the point of excitation; it seems as if the parts on either side of the irritated portion rise up and leave the furrow between them. The rising up of the irritated fibres like a thick cord, as Erb describes it, does not occur.

Smaller muscles, the interossei for instance, contract as a whole, and the contraction lasts for some time after the excitation has ceased. The length of time which the first described groove persists is something remarkable, and I have repeatedly seen it last from twenty to forty seconds. All of the voluntary muscles which were examined clearly showed this phenomenon, some to a greater, others to a lesser extent.

The *electrical examination* was made with great care and each experiment frequently repeated. Erb's cases were taken as guides.

*Motor nerves, faradic excitability.* Erb's results were fully corroborated. The experiments were performed over the accessory, ulnar and peroneal nerves. Very weak currents that, is, the minimum amount of current requisite to produce a contraction, brought about contractions which were not prolonged. Strong

currents produced contractions lasting from fifteen to twenty-six seconds after removal of the current. Single opening shocks never, even with the strongest currents, produce any prolonged contractions, but only short quick ones.

The *galvanic excitability* of the nerves showed nothing abnormal; tonic prolonged contractions of the muscles, as seen in consequence of their direct excitation, do not occur. Neither is there any reversal of the normal formula.

*Muscles.* *Faradic excitability* is very marked, minimum currents produced normal contractions, without any prolongation. Strong excitation produces contractions which last from twenty to thirty seconds after the current is broken. Single opening shocks uniformly produce only a short quick contraction, no matter how strong a current is employed.

*Galvanic excitability* was also found well pronounced. The muscles are very easily excited. They react only to closure contraction. The excitation is not always confined to the muscle acted upon, but seems to spread easily to contiguous muscles, so that entire groups are brought into contraction. Qualitatively what impressed me most was that the K.C.C. and An.C.C. do not seem to bear any fixed relationship to each other. This is very manifest. In most muscles the K.C.C. is obtained first and is followed at once, with only a very slight increase of current, by the An.C.C., but in some muscles they are both obtained with one and the same strength of current, and in others again the An.C.C. precedes the K.C.C. Besides this, the slowness with which the contractions take place and their long persistence is very noticeable. With fairly strong currents this slowness of contraction is shown by the gradual appearance of a deep groove in the muscle, which, if the electrode is moved along, can be moved with the electrode. If now the current be broken, the long persistence of the contraction becomes apparent, and this persistence is always in direct proportion to the strength of the current employed.

It is hardly necessary to give the figures as obtained from the various electrical tests; they do not differ materially from Erb's. *Rhythmical wave-like or undulatory contractions*, as described by Erb, I have at no time been able to obtain; but what I have repeatedly seen, and this has been verified by others who were thoroughly impartial, was a tonic contraction of the muscular groups, those at the kathode becoming first affected and finally those at the anode becoming involved, until the entire extremity was in a state of tonic contraction.

This successive implication of the various groups was very evident with different strengths of the current varying from 10 to 20 m.a. The stronger the current the more marked were the contractions. In the arm the kathodic-anodic contraction was always very plain. In the thigh the direction was not so evident.

The nerves of the arm, the radial, ulnar, and median, seemed to be particularly well adapted for the study of the difference between direct and indirect excitation. One and the same current in the

first case always producing short quick contractions and in the second slow, tonic, lasting ones.

All of the changes above described were found to be present in every muscle examined; some muscles, however, showed them more, others less distinctly. They were at all times most marked in the upper extremities.

The patient remained under observation until Nov. 5th, when he left New York to return home. Since then I have had two letters from him, a few sentences of which may be of interest.

In the first letter, dated November 7th, he says:

"I think the weather has something to do with my case. When I left New York, I felt all right. It was, as you know, a fine day. Well, a little after dark I got very restless; my legs began to ache and got very stiff, and when I got off the train at Watertown it was raining, so you see it must have been the change of atmosphere that affected me, and it always does. I forgot to tell you, doctor, that I had some trouble when born. I was delivered with instruments." The next letter is dated December 12th. In this he says: "I always feel better in winter; I will tell you when I begin to grow worse; it is generally about the breaking up of winter. We live up-stairs, and sometimes I come up on the jump, and perhaps inside of half an hour it will be all I can do to get up at all."

Dr. Willy Meyer was kind enough to excise a piece of the right quadriceps femoris muscle. The results of the microscopic examination are given below.

#### REMARKS.

There can be no doubt whatever that in this case the diagnosis of Thomsen's disease is correct. In all important points it coincides entirely with the disease as described by Thomsen himself. The only characteristic which is wanting is that of heredity, but it is not possible even here to say that there was no hereditary influence, as the family history is a very unsatisfactory one. The disease in this patient was probably congenital; certainly it has existed as long as he can remember. The myotonic affection, consisting of the peculiar inhibition of motion after a long rest, the slowness and stiffness in executing movements on account of tonic contraction of the muscles after any forced movement, was clearly present. The hardening and contracturing of the muscles after any sudden violent motion; the gradual return to a normal condition after the voluntary movements have been executed several times; the influence of psychical emotion,



of change of temperature, etc., are all perfectly characteristic of the affection. The athletic development of the muscles without any muscular tension during passive motion, and the absence of any other disorder, particularly the absence of any affection of the central nervous system, are all very important. Then, also, the objective examination showed a condition corresponding almost exactly to that found by Erb in his three cases. That the rythmical wave-like contractions, as described by Erb, were not found in this case is unfortunate, as it would have been interesting to corroborate Erb in this particular. Their absence, however, does not in any way invalidate the diagnosis, and it is possible that, if the patient had been under observation for a longer period of time, they might have been seen at some time or other. These undulatory contractions are so interesting that it will not be amiss to cite what Erb says regarding them :

“ If a ‘ large ’ electrode be placed upon the nape of the neck (or upon the sternum), a ‘ medium ’ one upon the palm of the hand, there occurs, with sixteen to eighteen cells, an equable tonic tension of all the muscles of the arm. After short stabile action, or after one or two pole changes, we now see from the lower ends of the flexors of the fingers (above the wrist-joint, volar surface) wave-like contractions take place in rhythmicâl order ; and these wave-like contractions move upwards when the kathode, downwards when the anode is in the hand ; therefore, they come from the kathodal side and move towards the anodal side. The single waves follow each other (like the waves of water produced by a falling stone), first with about a second interval (sometimes faster, sometimes slower, this is different on different days), and can be followed up to about the middle of the forearm ; then they become slower and disappear after a time. Increase of the current strength may produce them again. The electrodes during this remain perfectly quiet and unmoved.” In speaking of this phenomenon as it occurred in his third case, Erb says :

“ The best places of application are, for the flexors of the

forearm: palm of the hand or volar surface of the wrist-joint and nape of the neck. For the vastus internus, next to the patella and neck. For the gastrocnemius, tendon of the muscle and neck. The amount of current requisite for the production of the phenomenon varies from six to twenty m.a."

The points in the objective examination which are important are: The long persistence of the mechanical and electrical contractions of the muscles. The slowness and tonic character of the contraction and the difference found between the reactions of muscles and nerves. These differences were briefly as follows: Motor nerves show a normal mechanical excitability. To the faradic current they present qualitatively and quantitatively, with the exception of the prolongations of contractions to the strong currents, normal reactions. The galvanic contractions were short and quick, and not prolonged.

The muscles show an exaggerated mechanical excitability. Their galvanic and faradic excitability is quantitatively and qualitatively changed. Their contractions are slow, tonic, and prolonged.

For the various changes found in the muscles, Erb proposes the name "myotonic reaction," and says that henceforth all that will be necessary in order to objectively diagnose Thomsen's disease will be a few blows with a percussion hammer and a few closures of the galvanic current with the anode and kathode, and that by this simple examination simulation of this disease can always be detected.

*Microscopical Examination of Muscle.*—The peculiar electrical and mechanical reactions obtained in the foregoing examination seem to point directly to an affection of the muscles themselves, and indicate the necessity of their careful microscopical examination. Others, also reasoning in same manner, have examined pieces of muscles from myotonia congenita, Ponfick, Petrone, Jacusiel-Grawitz, Knud Pontoppidan, Rieder, and finally Erb, but all of them, with the exception of Erb, with purely negative results. Erb's microscopical examination of three cases re-

vealed so much that was new that the results in my case, which differ so little clinically from those of Erb, were awaited with considerable interest. I may say right here at the outset that all the points seen by Erb were substantially corroborated by my examination, but still more was seen, and this seems to me, as will be shown, to be of particular importance for the explanation of many of the phenomena seen clinically.

The specimens were obtained from a piece of the quadriceps femoris muscle, which was removed from the pa-



FIG. 1.—Normal muscle. Quadriceps femoris. Transverse section.  $\times 300$ .

tient under antiseptic precautions, and with the use of an Esmarch bandage. The fresh piece of muscle was dropped immediately after excision into a one-half-per-cent solution of chromic acid and left there until sufficiently hardened. It was then transferred into strong alcohol, imbedded in celloidin and cut both transversely and longitudinally. The sections thus obtained were then stained, some of them in an ammoniacal solution of carmine, and others in a one-half-per-cent solution of chloride of gold. Thereupon the specimens were mounted in glycerin. It is well here to lay stress upon this fact, as the illustrations in Erb's

monograph, repeatedly referred to above, were taken from specimens mounted in Canada balsam, and to this circumstance I am inclined to attribute the lack of details which is apparent in those drawings, and which differs so markedly from that which I have seen. The appearances in my own specimens under a comparatively low power (300 diam.) were so striking that the necessity of comparing them with sections of a normal muscle at once became

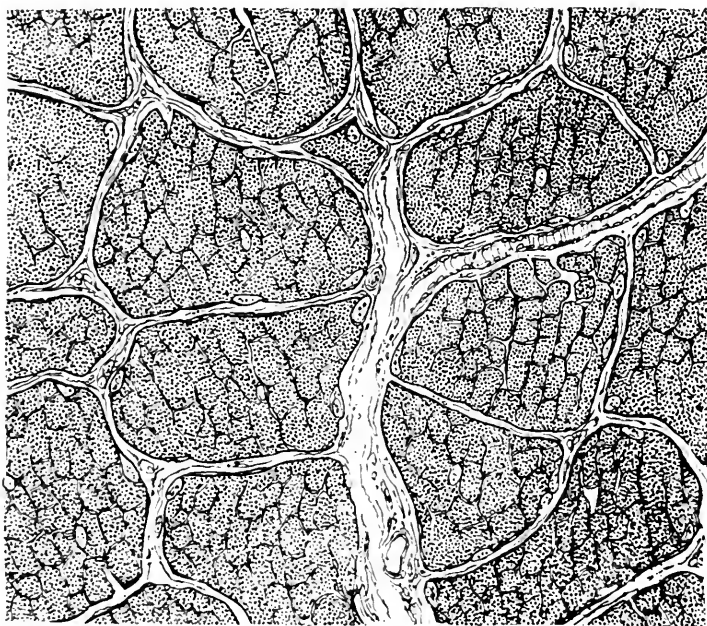


FIG. 2.—Myotonia congenita. Quadriceps femoris. Transverse section.  $\times 300$ .

apparent. The piece of muscle used for comparison was excised in the same manner, from the same locality, and from a healthy man of nearly the same age and size as was the diseased piece. It was then also placed at once in a one-half-per-cent solution of chromic acid and, in short, treated and mounted in the same manner as described above. Considerable time and care were necessary in order to obtain a suitable subject from whom to take this piece, but this was done purposely, as I feel sure that muscle taken from

a corpse would have been considered, and quite justly I admit, as worthless for the purposes of comparison and of drawing definite conclusions. Transverse sections of the affected muscles revealed with 300 diam. the following facts, substantially in agreement with the statements and conclusions of Erb.

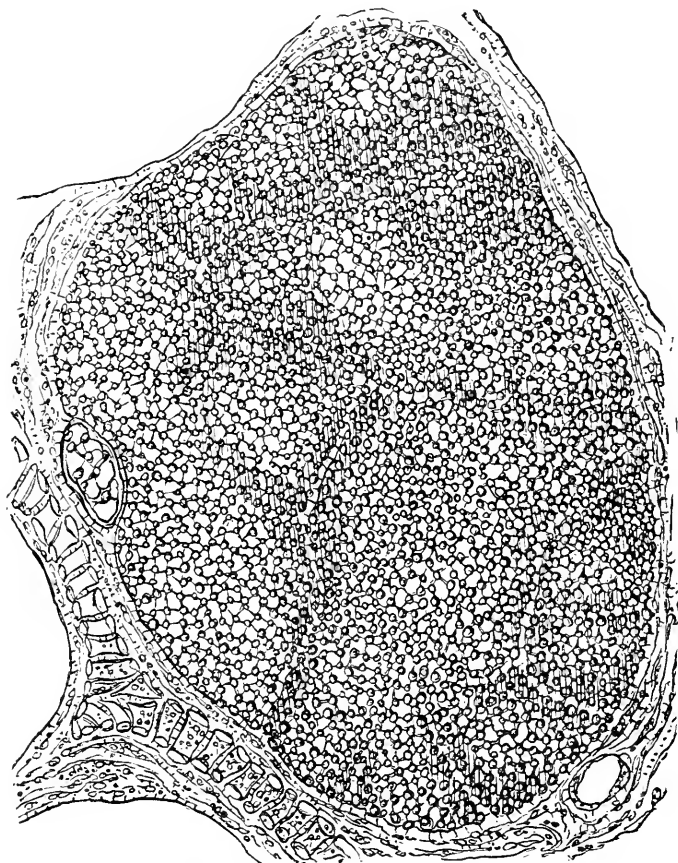


FIG. 3.—Normal muscle-fibre. Quadriceps femoris. Transverse section.  $\times 1200$ .

1st. The muscle fibres were about double the size of those in normal muscle, or perhaps on an average even larger. The illustrations of Erb's two cases show an increase in the size of the muscle fibres to the threefold or fourfold. My case was evidently, from a microscopical

point of view, not so marked as those of Erb. The fibres were also found to differ in shape from the normal, being more circular with somewhat rounded corners, and not polygonal as if from mutual pressure.

2d. The nuclei of the muscle-fibres were distinctly augmented in number, though also not as markedly as in Erb's cases. At the periphery of some of the fibres entire chains of nuclei could be traced, others showed scattered nuclei even in the middle of the fibre, a feature which is of comparatively rare occurrence in normal muscle.

3d. The connective tissue around the muscle-bundles (external perimysium), and that around the single fibres (internal perimysium), was found distinctly augmented. The internal perimysium, however, not to such a degree as in Erb's cases, since here and there it did not exceed that found in the normal condition. As normally, the external perimysium carried arteries and veins, the internal only capillaries. In several specimens I met with medullated nerve-fibres in the internal perimysium, and have also seen motor end plates at the periphery of some of the fibres, without finding any deviation in the course, number, or terminal arrangements of the nerve apparatus from the normal condition.

The peculiar formation of vacuoles in the muscle-fibres, as seen by Erb in two of his cases, was carefully and repeatedly sought for, but not a single specimen showed anything which could have been taken for them.

Transverse sections of the normal muscle with the same power exhibit marked differences in the interior of the various muscle-fibres. In the first place almost every fibre shows narrow angular slits, sometimes more, sometimes less numerous, which correspond to the embryonic sarcomeroplasts (Margo), which have entered into the construction of the single fibre. The entire surface of the fibre appears dotted, these dots corresponding, as is well known, to the sarcous elements of Bowman. The dots are either uniformly scattered or are arranged in rows, the latter condition obviously corresponding to a slightly oblique sec-

tion of the fibre. In some fibres also we see the sarcous elements closely packed together and coarse in appearance, in others they are minute, scarcely perceptible, and are widely separated from each other, thus rendering the

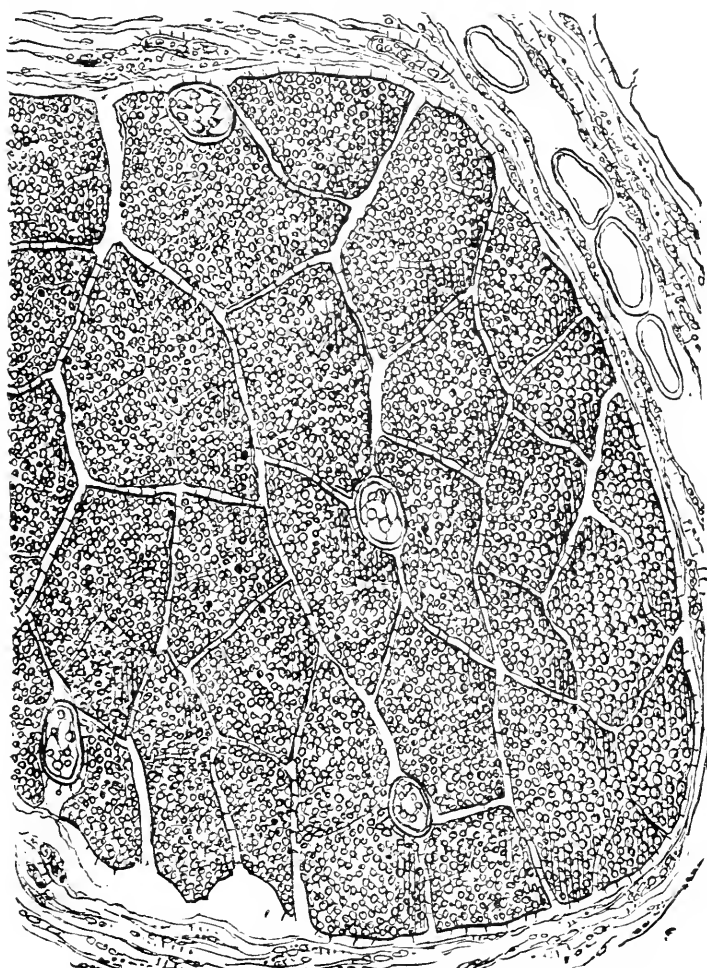


FIG. 4. —Myotonia congenita. Quadriceps femoris. Transverse section of a muscle fibre.  $\times 1200$ .

appearance of the entire fibre almost homogeneous. We even see these variations in one and the same fibre, it being in part coarsely, in part finely granular. Differences in the arrangement of the sarcous elements are known to exist

in accordance with the degree of contraction or extension of the muscle-fibre. When the muscle is completely at rest, we find the sarcous elements placed at regular intervals and exhibiting a certain size; this, however, varying in different muscles and individuals. During contractions the size of each individual sarcous element will increase and the interstices between them will decrease. In the state of extension, however, the single sarcous elements will decrease in size and the interstices between them become widened.

If now a piece of muscle be removed from the living body and at once placed into a solution of chromic acid, the state of contraction of at least a number of fibres, caused by the abrupt interruption of their continuity and by their exposure to the comparatively cool air, will be fixed by the chromic acid; hence we are justified in expecting to find, at any rate, a number of fibres in this state of contraction. Furthermore, we are justified in expecting to find various degrees of contraction in one and the same fibre. That our expectations are realized practically may be seen by reference to Fig. 1.

The difference in the interior structure of the single muscle-fibres from the case of myotonia congenita, as compared with that of normal muscle, is exceedingly well marked in transverse section. In the first place, nearly every fibre is distinctly divided into angular fields, varying in size, the same as are also seen in normal muscle, where, however, they are but faintly indicated (Cohnheim's fields). This splitting up of the fibre can in some places be traced into extremely minute fields, and in many instances is marked to such a degree that comparatively wide gaps are seen between the angular fields. Here and there also entire fields have dropped out, leaving large gaps behind. This latter feature is not infrequently seen along the periphery of some of the fibres, particularly in such cases where the fibre has been mechanically dragged out from its contiguity with the adjacent fibres. This feature is also alluded to by Erb.

The sarcous elements are also seen to be much more



uniform in size and arrangement than in the normal muscle, this being probably caused by the more general contraction of the muscular fibres (see Fig. 2), or by the uniformly smaller size of the sarcous elements, as mentioned later. Transverse sections of the normal muscle, when viewed with high power (1,200 diam.), show groups of sarcous elements, faintly separated from their neighbors by slight

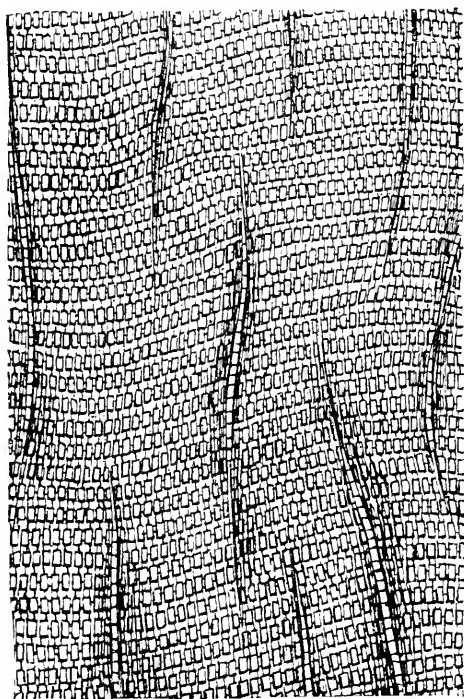


FIG. 5.—Segment of a normal muscle-fibre. Quadriceps femoris. Longitudinal section.  
X 1200.

interstices. The individual sarcous elements vary slightly in size and, especially in some of the muscle-fibres which apparently are not in a state of intense contraction, are seen to be distinctly connected with the adjacent elements by means of conical threads. These threads also traverse the interstices between the groups of the sarcous elements, and this connection is so well marked that in the entire field of a fibre not a single interruption between in-

dividual sarcous elements and groups of elements is visible.

Muscle-fibres which are evidently in a state of intense contraction show the sarcous elements lying close against each other, so that even when the short threads cannot be made out, a direct continuity is seen to be present by the immediate contact of the sarcous elements between themselves. Whenever a nucleus is seen at the periphery of a fibre, its contour is likewise seen connected with the adjacent sarcous elements by means of these delicate threads.

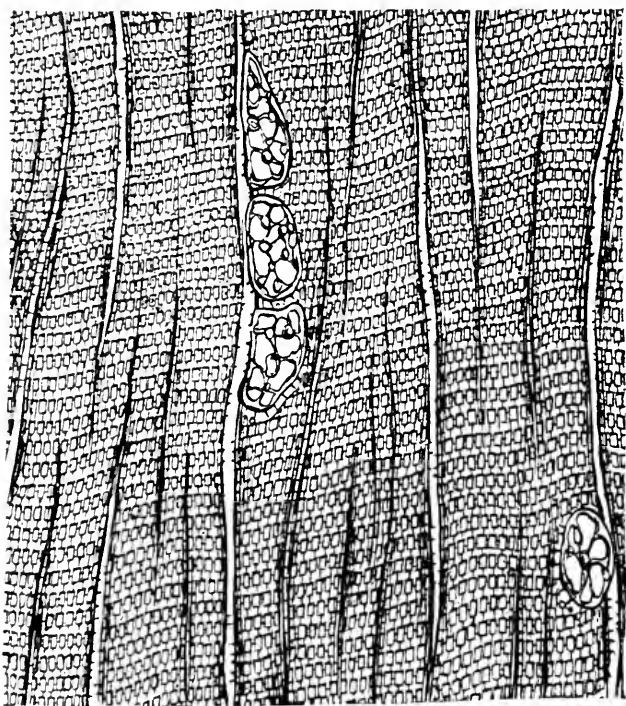


FIG. 6.—Segment of a muscle-fibre from myotonia congenita. Quadriceps femoris. Longitudinal section.  $\times 1200$ .

Furthermore, wherever there are motor plates at the periphery of a fibre, a continuity is also established between them and the neighboring sarcous elements (Fig. 3). Chloride of gold solution stains the sarcous elements and their connecting threads a violet color, thus rendering them more distinctly visible.

Transverse sections from the muscle affected with myotonia congenita, when regarded with the same power (1,200), show almost everywhere a lack of continuity between the groups of sarcous elements. This feature is most conspicuous along the splits and gaps spoken of above; secondary smaller splits, which traverse the larger groups of sarcous elements, show here and there only a few connecting threads.

The sarcous elements within the smallest fields are closely packed together, so that a mutual contiguity is established without the aid of intervening threads, and signifies, according to the statements made above, a high degree of contraction within a limited number of sarcous elements, and an interruption of continuity between smaller groups of sarcous elements (Fig. 4).

Longitudinal sections of normal muscle, viewed with high power (1,200), show the well-known variations in the arrangement of the rows of sarcous elements, down to the splitting up of the entire fibre into extremely delicate fibrillæ (Schwann's diagram). Within the fibres, slits, as is well known, exist, corresponding to the construction of the entire fibres from a number of embryonic sarcoplasts. Whatever be the arrangement of the rows of sarcous elements, these are invariably seen to be connected with each other by means of delicate threads in a longitudinal as well as in a transverse direction. Such connections are seen even where the sarcous elements form a continuous longitudinal line and the connections are traceable throughout all slits caused by sarcoplasts (see Fig. 5). Longitudinal sections of fibres from the abnormal muscle, with the same power, differ from the normal muscle by the presence of very marked slits, also corresponding to the boundaries of the sarcoplasts, in which, however, no connections are visible.

Particularly in these sections it becomes evident by comparison that all the sarcous elements are markedly smaller in the abnormal than in the normal muscle.

## CONCLUSIONS.

Clinically, Thomsen's disease appears to be an affection of the muscles alone, and microscopically this probability is only confirmed. It seems also as if the result of the microscopical examination were capable of casting some light upon the intricate nature of this peculiar affection. There can be no doubt that this disease is a congenital malformation of the muscular fibres, as is proven by the augmented size of each individual fibre, and by the increase in the number of their nuclei and of the perimysium.

It is obvious that a considerably larger number of embryonic sarcoplasts must have entered into the construction of each individual fibre than is the case in normal development. Such a muscle necessarily has a considerably larger number of sarcous elements, or which is synonymous, more contractile matter than a normal muscle, and that, therefore, the contraction of such a muscle is more liable to become exaggerated, as it were; or, in other words, to become tetanic. This is plainly seen in the specimens obtained from my case, where clusters of sarcous elements are seen to aggregate to a close contiguity. In normal muscle, the motor nerves are known to terminate in the form of plates beneath the sarcolemma, but upon the surface of the muscle-fibre. The continuity between the motor end-plate and the adjacent sarcous elements is established by delicate threads, and the continuity throughout the entire muscle-fibre is preserved by such filaments interconnecting all the sarcous elements in every direction, the interstices between the embryonic sarcoplasts not making any exception to this rule. Thus we conceive that the nerve-impulse, whatever this may prove to be, is transmitted from the motor nerve into the terminal plate, thence into the adjacent sarcous elements, and finally into all contractile particles of a muscle in a direct way, namely, by means of the connecting threads. In Thomsen's disease, the motor nerves and motor end-plates do not show any deviations from the normal. The

nerve-impulse, therefore, is transmitted into the muscle-fibre in the same manner as in the normal condition.

The result of this reception of impulse will be a contraction which, especially after a certain rest, will be a hypercontraction, or rather tetanus. This tetanus leads to an agglomeration of a certain number of sarcous elements, with a break in the continuity of the contracted clusters, as can be plainly seen under the microscope. In consequence of this tetanus, the nerve-influence is inhibited for so long as the tetanus lasts. After the lapse of a few seconds, the tetanic contraction will subside, the continuity between the hitherto separated groups of sarcous elements will become re-established, and the propagation of nerve-influence again rendered possible. If now in the light of our microscopical revelations, and in consideration of the above theory, we reconsider the objective symptoms found in Thomsen's disease, we are able to understand the production of a great many of them. That the muscle becomes tetanic under the influence of the will has been explained, that mechanical and electrical stimuli applied to the muscles themselves produce a prolonged contraction is also understood, but how it is that stimuli applied to the nerve do not have the same action as those applied to the muscle is not so clear. This much is certain, that the cause for this variation must be sought in some change in the nerves themselves, and not in the muscles, probably a change in their molecular arrangement, for microscopically the nerve terminations appear normal, and it is after all possible that later observations may discover changes either in the peripheral or central nervous system, which will take this peculiar affection out of the domain of primary muscular disorders to which it now appears to belong.

## THE HEAT-CENTRE IN THE BRAIN.

BY ISAAC OTT, M.D.

**T**WENTY years ago it was announced by Tscheschin that a heat-centre existed above the pons Varolii in the brain. Prof. H. C. Wood, in his large work on fever, has also by numerous experiments arrived at the same conclusion regarding the presence of a cerebral heat-centre. Richet<sup>1</sup> also discovered that the anterior part of the brain had an influence on the temperature of the body. The position of this centre was not located till I published a communication,<sup>2</sup> April 2d, 1884, stating that it was about the corpora striata. Drs. Aronsohn and Sachs<sup>3</sup> in December, 1884, discovered that puncture in the brain caused an increase of the temperature of the body. On July 4th, 1885, I published a preliminary communication,<sup>4</sup> stating that the heat-centre could be localized at the anterior inner end of the optic thalami and that the increase of temperature was due to increased production of heat. On Oct. 29th, 1885, Drs. Sachs and Aronsohn<sup>5</sup> published a paper stating that the heat centres were more accurately localized in the nodus corsorius and the tissues about the corpus striatum; and that the increase of temperature was due to increased heat-production. In this paper I propose to give the experiments upon which the preliminary note was based.

Forty experiments were made upon rabbits. The ani-

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<sup>1</sup> Bulletins de la Société de Biologie, March 29th, 1884.

<sup>2</sup> Journal of Nervous Diseases, April, 1884.

<sup>3</sup> Deutsche Medicinische Wochenschrift, No. 51, 1882.

<sup>4</sup> Philadelphia Medical News, July, 1885.

<sup>5</sup> Pflüger's Archiv.

mal was bound down, the scalp divided and the skull trephined, the dura mater divided, and punctures made through the brain by means of a small spear-pointed instrument two millimetres in diameter at the point and the remaining part one millimetre in thickness. The temperature-variations were noted by means of a rectal thermometer. The animal during this time was permitted to run about the laboratory. Punctures were also made upwards through the base of the brain by means of a stiff probe which passed through the skull by pressure. After all these punctures the animal was killed, the brain removed, hardened in alcohol, and sections made. To estimate heat-production, d'Arsonval's copper-calorimeter, surrounded by means of felt and feathers, was employed. The temperature of the calorimeter was noted by means of a thermometer inserted into the water contained in its closed cavity. Another thermometer inserted into the exit tube gave the temperature of the air aspirated from the calorimeter by means of the large meter of the Voit-respiration apparatus, which is run by a water-wheel. The temperature of the air of the room was also noted. The calorimeter was used when its temperature was a few degrees below that of the air. The large meter registered the amount of air passing through the meter. The calculations were made in the manner detailed in Wood's work on fever. All thermometers were corrected before the estimates were made. If the appended experiments are examined, it will be seen that at the anterior inner end of the optic thalamus a puncture causes a great increase of temperature in Exp. 12, almost 7° F. in an hour. That this rise of temperature continues on the following day is seen in several experiments, as in Exp. 8.

The calorimeter experiments show that the increments of temperature are due to increased production of heat and not to diminished dissipation.

In Exp. 16, the increase was 11.13 units; in Exp. 17, 10.49 units. In the calorimeter experiments, a great increase of temperature was not obtained, otherwise the units of heat-production would be much greater. Many

other calorimetrical experiments were made, but I do not think it necessary to publish them, as they are only confirmatory of those noted. Electric irritation by means of Du Bois-Reymond's inductorium caused an increase of temperature. It was also found that Fairchild's trypsin when dissolved in water with a small quantity of bicarbonate of soda and filtered, that the filtrate injected by the jugular caused a rise of temperature of four degrees in the rabbit.

These experiments prove that at the anterior inner end of the optic thalami a puncture causes an increase of temperature due to increased heat-production. Fig. A—1 shows about the point that the puncture should be made to cause the greatest increase of temperature; 2, is the corpus striatum; 3, optic thalamus; 4, corpora quadri-

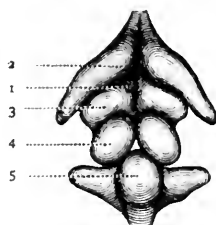


FIG. A.

gemina; 5, cerebellum. The cut was drawn by my student, Mr. Carter, from the brain of a rabbit.

Experiments made during last summer show that the tissues between the optic thalami and corpora striata along the median line also cause an increase of temperature, especially at the point which Schiff has pointed out as causing upon injury in rabbits a peculiar cry.

We have here an artificial fever due entirely to nervous disturbance, and not to any poisons circulating in the blood. The rise of  $7^{\circ}$  F. in an hour shows that the nervous system plays a very extended part in the phenomena of fever. As to the nature of these centres, all opinions are more or less conjectural. With our present information on other functions of the brain, the inference is that they are inhibitory in their nature.



I am indebted to my student, Mr. Peter J. Martin, for the calorimetical determinations.

*Exp. 1.*

Rabbit received a puncture through the anterior inner part of the right corpus striatum.

2.	P.M.,	.	.	.	.	.	.	.	.	T.
2.15	"	.	.	.	.	.	.	.	.	103 $\frac{3}{4}$
3.	"	.	.	.	.	.	.	.	.	104
4.	"	.	.	.	.	.	.	.	.	105 $\frac{1}{10}$
										104 $\frac{1}{2}$

Animal killed.

*Exp. 2.*

Rabbit. Lesion about the centre of the left optic thalamus.

1.45	P.M.,	.	.	.	.	.	.	.	T.
3.46	"	Puncture in thalamus,	.	.	.	.	.	.	102 $\frac{1}{2}$
5.	"	.	.	.	.	.	.	.	104 $\frac{7}{8}$
									105 $\frac{7}{8}$

*Exp. 3.*

Rabbit.

1.25	P.M.,	.	.	.	.	.	.	.	T.
2.45	"	Puncture in right corpus striatum to the base of the brain.	.	.	.	.	.	.	102 $\frac{1}{2}$
3.45	"	.	.	.	.	.	.	.	105 $\frac{1}{10}$
1.30	" next day	.	.	.	.	.	.	.	102 $\frac{1}{2}$

*Exp. 4.*

Rabbit.

2.10	P.M.,	.	.	.	.	.	.	.	T.
2.15	"	Puncture in left corpus striatum.	.	.	.	.	.	.	103 $\frac{1}{2}$
3.	"	.	.	.	.	.	.	.	105 $\frac{1}{2}$

*Exp. 5.*

Rabbit.

1.30	P.M.,	.	.	.	.	.	.	.	T.
1.35	"	Puncture at the anterior inner part of the optic thalamus just where it lies against the corpus striatum.	.	.	.	.	.	.	103 $\frac{1}{2}$

106 $\frac{1}{10}$

*Exp. 6.*

Rabbit.

1.35 P.M.,	.	.	.	.	.	.	.	T.
3.30 "	Puncture in anterior inner edge of thalamus,							103½
								105½

*Exp. 7.*

Rabbit.

2.07 P.M.,	.	.	.	.	.	.	.	T.
6.30 "	Edge of thalamus injured at its anterior part,							103¾
								105¾

*Exp. 8.*

Rabbit.

2.07 P.M.,	.	.	.	.	.	.	.	T.
3.	"	Puncture at anterior inner part of thalamus.						104½
6.30 "	.	.	.	.	.	.	.	108½
10.45 A.M., next day,	.	.	.	.	.	.	.	107½

*Exp. 9.*

Rabbit.

I	M.,	.	.	.	.	.	.	T.
1.40	"	Lesion at anterior inner part of thalamus.						103¾
4.	"	.	.	.	.	.	.	109½

*Exp. 10.*

Rabbit.

1.43 P.M.,	.	.	.	.	.	.	.	T.
1.44 "	Needle electrodes pushed into the brain; Du							105½
	Bois coil at 23 centimetres for ten seconds;							
	the points were in the corpus striatum and							
	the anterior inner edge of the thalamus.							
1.48 P.M.,	.	.	.	.	.	.	.	106
3.	"	.	.	.	.	.	.	107½

*Exp. 11.*

Rabbit.

3.40 P.M.,	.	.	.	.	.	.	.	T.
	Puncture in the optic thalamus.							103½
5.47 "								104½

*Exp. 12.*

Rabbit.

		T.
1.45 P.M.,	. . . . .	102 $\frac{1}{2}$
1.55 "	Puncture through the base of brain where optic thalami join.	
2.25 P.M.,	. . . . .	104 $\frac{1}{2}$
2.45 "	. . . . .	109 $\frac{5}{8}$
5.15 "	. . . . .	109 $\frac{1}{2}$

*Exp. 13.*

Rabbit.

		T.
1.55 P.M.,	. . . . .	103 $\frac{1}{2}$
	Puncture at base of brain through anterior end of optic thalamus.	
2.26 P.M.,	. . . . .	104 $\frac{2}{3}$

*Exp. 14.*

Rabbit.

		T.
3.10 P.M.,	. . . . .	103 $\frac{1}{2}$
3.15 "	Puncture through base of brain, through the an- terior ends of optic thalami.	
5.15 P.M.,	. . . . .	106

*Exp. 15.*

Rabbit.

		T.
1.20 P.M.,	. . . . .	103 $\frac{1}{2}$
1.30 "	Puncture through base of thalamus on left side.	
2.30 "	. . . . .	107

For every degree the calorimeter is below that of the air, a correction of  $\frac{9}{100}^{\circ}$  F. is made for the rise of temperature of the calorimeter during the experiment. It was found by several experiments that the calorimeter normally rose this much every hour, when a definite quantity of air was drawn through the calorimeter.

*Exp. 16.*

TIME.	A.T.	E.T.	C.T.	R.T.	M.
1.27	71.8	70.1	70.4	103 $\frac{1}{10}$	583,736
1.42	72.4	72.7	70.6		

1.57	72.5	72.7	70.8		
2.12	72.7	72.7	70.9		
2.27	72.8	72.7	71.	102½	588,116
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
	72.4	72.1	.6	.5 loss.	4,380
2.58	73.8	70.8	71.2	103½	
3.13	73.3	73.4	71.4		
3.28	73.3	72.7	71.5		
3.43	73.3	72.7	71.6		
3.58	73.4	73.4	71.8	106½	592,986
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
	73.5	72.6	.6	3½ gain.	4,870
Wt. 3.90 lbs.			Lesion ant. part of thalamus.		

$$V + (V \times t' \times .002035) = V'$$

$$V' = 4380 \text{ litres} \times 61.028 = 26730.26 \div 1728 = 154.6 \text{ cubic feet.}$$

$$t' = 72.1^\circ - 32^\circ = 40.1 \times .002035 = .0816.$$

$$V + .0816 V = 154.6 \text{ cubic feet.}$$

$$V = \frac{154.6}{1.0816} = 142.9 \text{ cu. ft. at } 32^\circ \text{ F.}$$

$$W = 142.9 \times .08073 = 11.55 \text{ lbs.} = \text{weight of air.}$$

$$Q = w \times t \times \text{sp. h.}$$

$$11.55 \times .3 \times .2374 = .820 = \text{heat from air.}$$

$$41.72 \times (.6 - .16) = 18.356 = \text{heat to calorimeter.}$$

$$17.536 = \text{heat dissipation.}$$

#### *After Operation.*

$$V' = 4870 \text{ litres} \times 61.028 \text{ cu. in.} = 29722.63 \div 1728 = 172.0 \text{ cu. ft.}$$

$$t' = 72.6 - 32 = 40.6 \times .002035 = .0826.$$

$$V + .0826 V = 172.0 \text{ cu. ft.}$$

$$V = \frac{172.0}{1.0826} = 158.8 \text{ cu. ft. at } 32^\circ \text{ F.}$$

$$w = 158.8 \times .08023 = 12.8 \text{ lbs.} = \text{weight of air.}$$

$$Q = w \times t \times \text{sp. h.}$$

$$12.8 \times 9 \times .2374 = 2.720 = \text{heat taken from air.}$$

$$41.72 \times (.6 - .15) = 18.774 = \text{heat to calorimeter.}$$

$$16.050 = \text{heat dissipation.}$$

*Heat Production.*

$Q = w + t + \text{sp. h.}$  W. 3.9 lbs.

Loss of temperature per hour,  $.5^{\circ}$ .

$3.9 + .5 + .83 = 1.615 = \text{heat from reserve.}$

$17.536 = \text{heat dissipation.}$

---

$15.921 = \text{hourly production.}$

*After Operation.*

Gain of temperature per hour,  $3.4^{\circ}$ .

$3.9 + 3.4 + .83 = 11.00 = \text{heat to reserve.}$

$16.05 = \text{heat dissipation.}$

---

$27.05 = \text{hourly production.}$

*Exp. 17.*

TIME.	A.T.	E.T.	C.T.	R.T.	M.
1.35	80.9	76.4	76.5	$102\frac{2}{3}$	628,846
1.50	81.3	78.8	76.8		
2.05	81.6	79.1	77.		
2.20	81.7	79.1	77.1		
2.35	81.8	79.1	77.3	$102\frac{2}{3}$	634,646
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
	81.4	78.5	.8	0	5,800
2.54	82.4	77.7	77.5	$102\frac{2}{3}$	
3.9	82.4	80.2	77.8		
3.24	82.4	80.2	78.		
3.39	82.5	80.	78.2		
3.54	82.5	80.	78.4	104	639,867
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
	82.4	79.6	.9	$1\frac{2}{3}$	5,221
Wt. 3.34 lbs.			Lesion in the optic heat-centre.		

$$V + (v \times t' \times .002035) = V'$$

$$V' = 5800 \text{ litres} \times 61.028 = 35396.24 \div 1728 = 204.0 \text{ cu. ft.}$$

$$t' = 78.5 - 32 = 46.5 \times .002035 = .0946.$$

$$V + .0946 V = 204.0 \text{ cu. ft.}$$

$$V = \frac{204.0}{1.0946} = 18.64 \text{ cu. ft. at } 32^{\circ} \text{ F.}$$

$$w = 18.64 \times .08073 = 15.0 \text{ lbs.} = \text{weight of air.}$$

$$Q = w \times t \times \text{sp. h.}$$

$$15.0 \times 2.9 \times .2374 = 11.40 = \text{taken from air.}$$

$$41.72 \times (.8 - .36) = 18.35 = \text{heat to calorimeter.}$$

---


$$6.95 = \text{heat dissipation.}$$

*After Operation.*

$$V' = 5221 \text{ litres} \times 61.028 = 31856.51 \div 1728 = 184.3 \text{ cu. ft.}$$

$$t' = 79.6 - 32 = 47.6 \times .002035 = .0968.$$

$$V + .0968 V = 184.3 \text{ cu. ft.}$$

$$V = \frac{184.3}{1.0968} = 16.80 \text{ cu. ft. at } 32^\circ \text{ F.}$$

$$W = 16.80 \times .08073 = 13.5 \text{ lbs.} = \text{weight of air.}$$

$$Q = w \times t \times \text{sp. h.}$$

$$13.5 \times 2.8 \times .2374 = 8.960 = \text{taken from air.}$$

$$41.72 \times (.9 - .36) = 22.528 = \text{heat to calorimeter.}$$

---


$$13.568 = \text{heat dissipation.}$$

*Heat Production.*

$$Q = w \times t \times \text{sp. h.} \quad W. 3.34 \text{ lbs.}$$

No change of temperature.

$$\text{Heat dissipation} = 6.95 = \text{hourly production.}$$

*After Operation.*

Gain of temperature per hour,  $1.4^\circ \text{ F.}$

$$3.34 \times 1.4^\circ \times 83 = 3.881 = \text{added to reserve.}$$

$$13.568 = \text{heat dissipation.}$$

---


$$17.449 = \text{heat production.}$$

*Exp. —*

TIME.	A.T.	E.T.	C.T.	R.T.	M.
1.25	72.6	71.6	72.2	103 $\frac{1}{2}$	617,667
1.40	72.7	73.7	72.4		
1.55	72.9	74.4	72.5		
2.10	73.2	74.4	72.7		
2.25	73.5	74.9	72.8	102 $\frac{3}{4}$	623,073
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
	72.7	73.8	.6	1 $\frac{1}{2}$ loss.	540.6

4.10	76.9	74	73.3	103 $\frac{1}{2}$	
4.25	77.4	74.9	73.5		
4.40	77.4	75.9	73.7		
4.55	77.8	75.9	73.9		
5.10	78.2	76.2	74.1	104 $\frac{3}{4}$	628,846
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
	77.5	75.4	.8	$\frac{1}{8}$ gain.	577.3

Wt. 3.35 lbs.

Lesion in corpus striat. and optic thalamus.

$$V + (v \times t \times .002035) = V'$$

$$V' = 5406 \text{ litres} \times 61.028 = 329551.2 \div 1728 = 190.6 \text{ cu. ft.}$$

$$t = 73.8 - 32 = 41.8 \times .002035 = .085.$$

$$V + .085V = 190.6 \text{ cu. ft.}$$

$$190.6$$

$$V = \frac{\quad}{1.085} = 179.7 \text{ cu. ft. of air at } 32^\circ \text{ F.}$$

$$1.085$$

$$w = 179.7 \times .08073 = 14.5 \text{ lbs. = weight of air.}$$

$$Q = w \times t \times \text{sp. h.}$$

$$14.5 \times 1.1 \times .2374 = 3.780 = \text{heat added to air.}$$

$$41.72 \times .6 = 25.032 = \text{heat to calorimeter.}$$

$$\underline{\quad\quad\quad} \\ 28.810 = \text{heat dissipation.}$$

*After Operation.*

$$V' = 577.3 \times 61.028 = 352131.5 \div 1728 = 203.8 \text{ cu. ft.}$$

$$t = 75.4 - 32 = 43.4 \times .002035 = .0883.$$

$$V + .0883V = 208.3 \text{ cu. ft.}$$

$$208.3$$

$$V = \frac{\quad}{1.0883} = 191.4 \text{ cu. ft. at } 32^\circ \text{ F.}$$

$$1.0883$$

$$W = 191.4 \times .08073 = 15.4 \text{ lbs. = weight of air.}$$

$$Q = w \times t \times \text{sp. h.}$$

$$15.4 \times 2.1 \times .2374 = 7.660 = \text{heat from air.}$$

$$41.72 \times (.8 - .31) = 20.442 = \text{heat to calorimeter.}$$

$$\underline{\quad\quad\quad} \\ 12.782 = \text{heat dissipation.}$$

*Heat Production.*

$$I = w + t + \text{sp. h.} \quad W = 3.35 \text{ lbs.}$$

$$\text{Loss of temperature per hour, } 1.2^\circ$$

$$\begin{array}{rcl}
 3.35 + 1.2 + .83 & = & 3.33 \text{ taken from reserve.} \\
 28.81 & = & \text{heat dissipation.} \\
 \hline
 25.48 & = & \text{hourly production.}
 \end{array}$$

*After Operation.*

$$\begin{array}{rcl}
 \text{Gain in temperature per hour, } 8^{\circ} & & \\
 3.35 + .8 + .83 & = & 2.22 = \text{added to reserve.} \\
 12.78 & = & \text{heat dissipation.} \\
 \hline
 14.98 & = & \text{hourly production.}
 \end{array}$$



## DIAGRAMS

TO ACCOMPANY DR. MITTENDORF'S PAPER ON "OPHTHAL-  
MOPLEGIA," P. 78 OF THIS VOLUME.

HENSEN & VOELCKER'S

Schematic arrangement of nuclei of 3d nerve.

LEFT.		RIGHT.	
1. Ciliary muscle	{ .	. }	Ophthalm.
2. Sphinct. of iris.	{ .	. }	interna.
3. Rectus intern.	{ .	. }	
4. Rect. sup.	{ .	. }	
5. Lev. palpebr. sup.	{ .	. }	Oph.
6. Rect. infer.	{ .	. }	externa.
7. Obliq. inf.	{ .	. }	
Trochlearis.	{ .	. }	
Abducens.	{ .	. }	

## KAHLER &amp; PICK'S

Schematic arrangement of nuclei of 3d nerve.

	LEFT.		RIGHT.	
1.	Ciliar. muscle . (accom.)		.	Ophth. int.
2.	Sphincter . iridis.		.	
5.	Lev. palp. sup.	3. . Rect. intern.	.	Ophth. externa.
6.	. Rect. sup.		.	
7.	. Obliq. inf.	4. . R. inf.	.	
	Trochlearis .		.	
	Abducens .		.	

## Clinical Cases.

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### A CYST OF THE PIA MATER IN A CHILD FIFTEEN MONTHS OLD.

BY L. EMMETT HOLT, A.M., M.D., OF NEW YORK.

M. S., aged fifteen months, an inmate of the New York Infant Asylum, came under my observation in August, 1885. The family history was tubercular, and a grandmother seemed to have been imbecile.

The child was born in the institution. The mother was a primipara, nineteen years of age. The child presented in the R. O. A. position, and was delivered after a natural labor of seven hours. The child never had any convulsions, nor any illness except sub-acute laryngitis. It was nursed by the mother, but was delicate and backward in its development. It had never walked alone, made no attempts to talk, and the nurse who had charge of the child during the later months thought its hearing was defective. This was brought out in response to inquiry after the child's death, but the physician's attention was never called to the fact.

The child had a peculiar habit of swaying its body backwards and forwards, which was continued much of the time during its waking hours. The child was taken with entero-colitis in the middle of August, developed broncho-pneumonia a few days later, and died after a two weeks' illness. Marked cerebral symptoms were present only during the last three days. These were nystagmus, muscular tremors, irregular movements of the hands and feet, irregular respirations, dilated pupils and opisthotonos. The child died of pulmonary œdema.

The autopsy was made twenty-one hours after death. The body was thin, but not emaciated.

The evidence of quite an extensive broncho-pneumonia was found in the lungs, and of a moderate enteritis in the intestines, but nothing of special interest except in the brain.

The brain was intensely congested, sinuses all distended with fluid blood, cerebro-spinal fluid greatly in excess; four ounces were collected. The pia was dull in appearance, but no exudation of lymph or pus was anywhere present, and no tubercles. After removal of the brain from the cranial cavity, a deep depression was

seen in the temporal lobe. It occupied the interior and upper portion of the lobe, encroaching on the first and second temporal convolutions. This cavity was conical in shape, with its apex directed inwards and slightly upwards; a horse chestnut could be laid in it easily. It measured one inch across its base and one and one-fourth inches in depth. Before removal, this had evidently been converted into a cyst by the pia stretching across its base, this had been lacerated in removal, allowing the fluid contents to escape. On floating the brain in water, the edges of the torn pia could be seen to meet across the depression.

At the bottom of the cavity the brain substance was firm and slightly pigmented. It was composed of gray matter and was covered with the thin cyst-wall.

A microscopical examination was made of the wall of the cyst



Cyst of the Pia Mater.

and the brain substance immediately beneath it. It showed the cyst-wall to be made up of the pia-mater greatly thickened. This connective-tissue growth compressed the cortex, but did not invade it below its superficial layers. The cells of the cortex were not altered appreciably. Here and there large-sized capillaries were visible. No cysts of hematin were seen. At one point was a granular mass which might be possibly pigment. The appearance of the connective tissue was old and well organized, such as might be expected in the wall of such a cyst. The exact location of the cyst is shown in the accompanying sketch made by Dr. Frank W. Olds.

Regarding the origin of the cyst, we are left completely in the dark by the clinical history. It will be seen that at no time during birth or in after-life did any symptoms present themselves pointing to a meningeal hemorrhage. If such is to be assumed as

the explanation, it must have occurred *in utero*. The slight degree of pigmentation at the bottom of the sac noted in the fresh specimen favors such an opinion. Further than this there was no evidence, pathological or otherwise, for regarding it as anything else than a simple cyst of the pia.

15 EAST FIFTY-FOURTH STREET, NEW YORK.

## REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILADELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF W. H. WALLACE, M.D., PHYSICIAN-  
IN-CHIEF, AND CHARLES K. MILLS, M.D., CONSULTING PHYSICIAN.

### *Three Cases of Epileptic Insanity.*

Reported by Dr. J. Chalmers Da Costa, Assistant Physician.

#### CASE IX.—*Pre-Epileptic Insanity.—Simulation of Epilepsy by the Same Patient.*

P. D— was admitted to the hospital early in November, 1885. He was born in Ireland, was 27 years old, married, a barber, and had a good common-school education. His family history was not clear, but no instances of heredity were discovered. He had a son about six years old who had had a fit.

He had been an epileptic for twenty years, and a hard drinker from boyhood. At first his attacks were rare, but became more frequent until he was obliged to give up his business in 1882. He became gloomy, irritable, and peculiar, which his wife attributed to remorse because he contracted gonorrhea from a prostitute. For three years before his admission to the hospital, he was depressed and weak-minded, with occasional outbreaks of violence, and he had many fits. When first in the hospital, he was extremely violent and destructive, talking with great rapidity, singing, jumping about, swearing frightfully, and attacking all who approached him. He refused food and did not sleep. He had a fierce and angry expression; his speech was incoherent, and he evidently labored under hallucinations of sight and hearing.

Nov. 11th, 1885.—He is violent, filthy, and destructive. He was fed with the nasal tube and went to sleep under hyoscine hydrobromate.

Nov. 14th.—The patient is remarkably improved. He began to eat voluntarily and was much less violent, though he still was confused and had several violent fits during the day.

Nov. 17th.—His mania is entirely gone; he eats and sleeps well, but is still confused.

Since that time he has frequently, but not invariably, before a fit been filthy in word and deed, malignant, dangerous, incoherent,

and destructive, often attacking people without cause. These attacks have lasted from a few hours to several days, and have ended with one or more fits, the number and violence of which seem to bear a direct relation to the intensity of the preceding mania. He has had attacks of violence which have not been observed to terminate in fits. This has been only when he went to bed maniacal and got up quiet, so that he probably had the seizure in the night. Sometimes his antecedent mania lasts for one week or more. One attack of violence lasted from Nov. 2d, 1885, to Nov. 14th; another from July 28th, 1886, to August 5th.

This case illustrates pre-epileptic insanity, the fit seeming to act as a safety valve, or rather as a throttle valve setting force into motion by being pulled out. It would seem as if nerve force were exploded on the mind, and was drawn from there to the muscles by Nature, thus converting a mental convulsion into a muscular one. Besides these transitory attacks, the patient has increasing dementia, more confusion, weakened will, memory, and judgment. His physical condition is good, and he shows no signs of syphilis or phthisis.

He occasionally simulated epilepsy for the purpose of getting tobacco; but he proved to be a poor actor, and his deception was easily discovered.

CASE X.—*Post-Epileptic Insanity.—Extreme Sexual Excitement and Aberration.*

R. S——, aged 30, was admitted to the Philadelphia Hospital in 1880. (The notes of his state on admission were destroyed by the fire of Feb., 1885. His family history was meagre and unsatisfactory; but none of his relatives were ever known to have been insane or epileptic.)

When he was 10 years old he fell from an ice wagon, and soon after this, fits set in. The only sickness he ever had was the measles when he was very young. It was noticed that on several occasions after having a seizure he was very violent. Once he tried to jump out of a window, and another time attacked his brother without any provocation. After a time, his mind showed marked and continued impairment, and he talked and acted absurdly at all times.

He is short and very stout, and has a peculiarly gross and coarse appearance. He has thick lips, and a lascivious smile. In his normal state he is quiet and tractable, and often helps in the ward. He is good-natured, and will try to kiss men. His memory for recent events is almost gone, and he has marked confusion of thoughts. He has never had any head injury so far as can be discovered.

About once a month he becomes depressed, and this depression is followed by violent fits, usually from one to four in number. These sometimes happen in quick succession, another fit coming on before the post-epileptic sleep ends (*status epilepticus*). At

other times the paroxysms are some hours apart. Between the fits and after them, he becomes very excitable. He shouts and sings, says he is "glorified and sanctified." He evidently has hallucinations of sight and hearing of a pleasant nature. He tries at these times to kiss every one who approaches him. He occasionally eats excrement and drinks urine. He practises sodomy and masturbates fiercely, and will scream out vile remarks without apparent shame. He has been observed to place his penis in his mouth. In order to do this he gets upon his back, elevates his thighs, and puts the perinæum on the stretch by pulling on the scrotum. His penis is not of abnormal size, but his back is peculiarly flexible. This vile state generally lasts for a day or two, and is accompanied by marked wakefulness. He then has a sound night's sleep, and in the morning has returned to his normal state. During the mania his eye-ground shows active congestion.

CASE XI.—*Post-Epileptic, Pre-Epileptic, and Replacing Mania.*

M. W——, born in Philadelphia, is 25 years of age, white, and a printer. His family history showed no cases of epilepsy, phthisis, or insanity. His father and mother are temperate, and he has three brothers and four sisters alive and well. He had never been addicted to the use of alcohol, and had never had any severe sickness. He had no signs of head injury, and no history of sun-stroke, syphilis, or masturbation.

When he was 8 years of age he had a severe fright from the house taking fire, and three months after this began to have attacks of "giddiness," in which he would cry out, fall, and remain unconscious for a time. After these he suffered with slight headache. At first he had two or three attacks in a month, but they increased in frequency until 1884, when he often had two a day. For some years these attacks were preceded by marked hallucinations of sight, the most constant object he saw being a shoe with a leg, which ran round and round until he lost consciousness. After a time a great change took place in the patient's disposition; he became dull and irritable, and imagined kindnesses were intended as injuries; he would also have outbursts of causeless anger, in which he occasionally tried to injure people. He once went into a jewelry store, broke the show-case and threw the watches and trinkets around, but made no attempt to steal anything. He became very egotistical, and would not tolerate the slightest contradiction. In 1884, he attacked a friend who, he said, owed him money, and was committed to the Philadelphia Hospital by order of the Court.

Examination showed his pupils to be equal and normal in size; they reacted to the light. His memory was beginning to fail; his emotions were unrestrained and his judgment was not acute; he was very egotistical. His urine and renal organs were normal; his lungs healthy, and his heart action, though weak, showed no murmur. He said that he felt "perfectly well."



On admission, he was having two fits a day, but under treatment they have diminished greatly. He has both diurnal and nocturnal epilepsy, and both grand mal and petit mal. For a time he had a stomach aura, but now a fit is preceded by a cold feeling running up the arms from his hands, he raises his arms over his head, gives a cry, and falls. His pupils dilate and he passes into a tetanic state which lasts from half a minute to a minute. Convulsive movements begin in the eyelids and face, and then the whole body is involved in the convulsion. This stage lasts for two or three minutes, and he then usually sleeps for an hour or two, and awakes with a slight headache, and a feeling of heaviness.

Besides his mild chronic dementia, he is beginning to labor under outbursts of acute maniacal excitement. On several occasions he has been observed to be more than usually excitable, demanding his release from "illegal confinement." He attacks people without any cause, breaks windows and doors, etc. After being several hours in this state, a fit usually occurs, perhaps more severe than common, and this relieves the mania.

Sometimes again after a fit he does not sleep, but gets up almost directly in a maniacal condition, and swears, fights, and breaks everything he can get at. This lasts sometimes for hours, and has continued for a whole day, until it is dissipated by sleep, either natural or induced by medicine. Again, he will pass some time without a fit, and then have a maniacal outbreak which will last an irregular time. While in this condition he is very dangerous. This also passes off in sleep.

This patient is an interesting one from several points of view. He shows three successive auras; the first, an hallucination of sight; the next, a stomach aura (which is far more common); and the third, a sensation from the hands and arms. He exhibits the beginning of mental deterioration, and illustrates true epileptic insanity in several forms: (1) pre-epileptic mania, which is stopped by a fit; (2) post-epileptic mania, which arises because of the absence of the post-convulsive sleep, and passes into sleep; (3) a mania taking the place of a fit, or what Spitzka has called the "psychical equivalent of a fit," and which, like the other forms, passes off by sleep.

## Periscope.

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PATHOLOGY (INCLUDING PATH. ANATOMY).

**On the Changes in Nerves and Spinal Cord after Amputations.** By C. FRIEDLAENDER and F. KRAUSE. (*Fortschritte der Medicin*, 1886, No. 23.)

Various changes in the spinal cord have been observed after amputations, consisting of atrophy of the gray substance both in the anterior and posterior horns, and of the white columns. No single lesion appears to be uniformly present. Authors hitherto have denied the existence of changes in the nerves or nerve roots from the stump to the spinal cord. Friedländer and Krause have recently examined the condition of both nerves and spinal cord in eight cases which died several years after amputations had been performed. Their results are as follows:

1. There are changes in the peripheral nerves traceable from the stump to the nerve roots, and these can be followed to the posterior spinal ganglion, but not into the posterior or anterior nerve root. The change is not a Wallerian degeneration, but is a simple atrophy. It involves as much as one-half of such a nerve trunk as the sciatic, many fibres in each bundle being affected. The individual fibres appear to have lost their medullary sheaths, and although a faintly tinged substance is left representing the axis cylinder, it is by no means certain that this is a normal axis cylinder. The nuclei of the sheath of Schwann are increased in number. The atrophied fibres have no double contour, do not stain with osmic acid, or with Weigert's hematoxylin, and stain only faintly with carmine or aniline-blue. This atrophy is to be found three months after amputation, but is more evident two or three years after. Since this atrophy is only to be found in the portion of the nerve which turns backward to the spinal ganglion, it must be admitted that the atrophic fibres are sensory in their function. The atrophy ceases at the posterior spinal ganglion, and no trace of it is to be found in the posterior nerve-root between the ganglion and the cord. The anterior-nerve root is free from any trace of atrophy. All the sensory nerve-fibres in a nerve from the stump are not involved. It is only a portion of these which are atrophied. The number seems to be about the same, whether the amputation is near the trunk or far from it. The authors put forward the hypothesis that the atrophied fibres are those which

come from the terminal bulbs and tactile corpuscles which are situated chiefly in the skin of the feet and hands. They think that the sensory fibres which are preserved are those which come from the sensory plexus in the skin. This implies that the tactile corpuscles and terminal bulbs are trophic organs for the fibres originating from them.

2. There are also changes in the spinal cord. (a) There was observed a diminution in the size of the posterior columns on the amputated side. When the leg was amputated, this was visible in the lowest part of the lumbar enlargement, and reached its maximum at the eighth dorsal segment, where the columns were only one-half the size of the opposite side. No atrophy of individual fibres was observed; there was simply a less number of normal fibres. No other columns of the cord were affected.

(b) There was observed a diminution in the number of cells in the gray matter, and a diminution in the size of the posterior horn. All the groups of cells in the anterior horn are not equally affected. The anterior median, antero-lateral, and central groups appear to be unaffected. The postero-lateral group is very markedly involved, being reduced to one-third or one-half the number of cells on the opposite side. Numerous careful observations bear out this statement, the numbers being given in the text. It is only the lower portion of the lumbar spinal cord in which this atrophy is seen, viz., that part from which the sacral nerves arise. The Clarke column of cells in the posterior median area of the gray substance was also found affected on the side of the amputation. The reduction in the size of this group was found to extend from the twelfth to the sixth dorsal segment. The number of cells in any single section was at least one-third less on the side of the amputation. When the amputation had been made in the upper extremity, the same changes were observed, excepting only those in the Clarke column which, as is well known, does not extend above the eighth cervical segment. The authors claim that this establishes the fact of an intimate connection between the Clarke column of cells and the sensory roots; also between the postero-lateral group of cells and the sensory roots.

3. A reduction in the number of fibres, but no appearance of atrophy in individual fibres, was observed in the posterior nerve-roots corresponding in degree with the atrophy in the posterior columns. A bibliography of the subject with critical comments is appended to the article.

M. A. S.

**Primary Degenerative Neuritis.** PROF. A. KAST.  
(*Deutsch. Arch. f. Kl. Med.*, Vol. 49, I., p. 41.)

Prof. Kast relates four interesting cases of various forms of neuritis, and in connection with each one of these has some valuable suggestions to offer.

CASE I.—Girl, aged 13, very mild angina follicularis; about two

months later paresis of accommodation; ataxia of upper and lower extremities. Marked disturbances of every form of sensibility; rheumatic pains, but nerves not painful to touch. Marked atrophy of muscles in keeping with general condition; no asymmetrical atrophies. Several months later, atrophy of interossei and of tongue; loss of faradic contractility; diminished galvanic excitability; deep reflexes absent; well-known bulbar symptoms super-added nine months after onset of disease, death from pneumonia. Autopsy: no changes in central nervous system (bulbar symptoms were due to peripheral nerve affection). Degeneration of various peripheral nerves—recurrent, hypoglossal, and various nerves of brachial plexus. (There are no illustrative plates.)

As this was a case with autopsy, it will be well to note the youthful age of this patient; furthermore, that such cases as these are much more than mere "painful paralyses." Apropos of this case, the author discusses the origin of the ataxia. He lays stress upon the fact that the inco-ordination is far in excess of the sensory disturbances. Referring to the researches of H. Tschierjew, he thinks it plausible to assume that the deficiency of the muscular sense is to be held accountable for the ataxia.

CASE II.—Typical case of alcoholic neuritis. Optic nerve first to be affected, various atrophic paralyses in all four extremities. Delirium tremens complicating croupous pneumonia. Patient was examined five months later.

The electrical reactions are given in great detail and are carefully discussed. Within the distribution of a single nerve some muscles showed normal reactions, others complete reaction of degeneration, and still others only partial R. D. Thus faradic stimulation of R. crural nerve produced contraction of sartorius and of all the divisions of the quadriceps femoris except the vastus internus, which failed to respond; the same was true of some other muscles. This difference in electrical behavior the author considers characteristic of peripheral neuritis rather than of poliomyelitis, for, as he justly argues, we could not imagine such changes to be due to a difference in the degeneration of various ganglion cells presiding over any one physiological group of muscles. Dr. Lloyd has recently insisted on very different points in differential diagnosis. The reader will observe that Dr. Lloyd<sup>1</sup> holds that the retention of electro-excitability in the nerves does not appear consistent with the idea of a neuritis, but rather with a slow cord lesion. "If this electric excitability can be preserved in a chronic neuritis, we must suppose a very slow interstitial inflammatory process which compresses some fibres and allows others to escape. But to think that this happens in the narrow calibre of a nerve-trunk during a prolonged period of inflammation seems to require some effort of imagination and credulity"—the exact opposite of Kast's views. We think Kast's argument entirely sound, in perfect agreement with his careful electrical examina-

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<sup>1</sup> See report of Philadelphia Neurological Society in this number.

tions. But the two authors have based their conclusions upon entirely different cases. No doubt in some cases Lloyd's views will prove correct. The entire subject needs further careful study. Possibly, too, when all the facts are known, we may find that electrical tests will not furnish points of differential diagnosis between neuritis and anterior poliomyelitis.

CASE III.—Female; æt. 23. Eleven days after confinement, septic fever lasting for nearly two months. At the end of this time, patient developed atrophic paralysis in the partial distribution of ulnar and median nerves, with some partial and some complete R. D. Violent pains in arms. Pain on pressure over nerve trunks; transient paresis and paræsthesiæ of legs, possibly septic endocarditis; complete recovery within little more than a year. Case is interesting as an instance of multiple neuritis developing in the wake of a tangibly infectious disease.

CASE IV.—Man; æt. 21. Was given an hypodermic injection of ether into the upper third of left forearm. In a few days patient developed typical musculo-spiral paralysis. Complete R. D. in entire distribution of the nerve. No very marked sensory symptoms, but considerable subjective pain. Recovery within a few months. This accident suggests a way in which neuritis might be studied experimentally.

B. S.

#### MENTAL PATHOLOGY.

**Cases of Microcephalon Caused by Psychical Influences during Pregnancy.** PROF. C. LOMBROSO. (*Arch. di Psichiatria, Scienza Penale ed Antropologia Criminale*. Vol. VII. Fasc. II. 1886.)

The first case reported by Dr. Lombroso is that of a microcephalic idiot, aged 7, whose mother, a healthy and fine contadina, aged 20 at the time the child was born, was frightened by a large ape in the first months of her pregnancy. The impression made was profound and remained for a long time. The father and sisters of the child were robust and healthy.

The second case is that of a youth of 20, exhibiting retarded development, looking not more than 15. His three brothers were healthy, although small of stature, as were both father and mother. He did not walk until 7. He did not make gestures or pronounce an intelligible syllable until he was 12. He showed great signs of fear and exhibited great cruelty to animals. His skull was asymmetrical and the cranial capacity much less than normal.

His mother suffered from a great fright some time between the third and sixth month of her pregnancy from seeing a soldier threatening to split the head of her husband with a sickle. From that time she was in a state of fear, with trembling, cramps in her members, without appetite, and a sense of cold throughout her body.

Prof. Lombroso thinks that psychical origin of an embryonal deformity is not at all inadmissible when one considers the persis-

tence of this opinion through centuries, and also that the modern theory of mental suggestion offers an explanation of this influence and shows the parallelism between powerful psychical impressions and physical modifications.

GRACE PECKHAM.

**Contracts with the Insane, Wills of the Insane, and Testamentary Capacity.** DR. CHARLES K. MILLS. (*Medical and Surgical Reporter*, Philadelphia, July, 1886.)

Dr. Mills, in a series of lectures on insanity, delivered at the University of Pennsylvania, thus refers to business, marriage, and other contracts with the insane.

"The validity of civil contracts made by the insane, like so many other questions pertaining to insanity, must be decided largely by the circumstances surrounding the particular case. While we have decisions and laws relating to matters of this kind, yet these are not so absolute, and are not rendered so closely after previous cases, but that doubts may arise in a particular case. The mere fact of the existence of insanity will not always invalidate a contract. The particular circumstances surrounding such a case must always be taken into consideration. If a merchant, although insane, made a contract, and if the party with whom the contract was made would be the loser if it was not fulfilled, the law in all probability would hold the firm to which the insane man belonged liable. In one of the cases I have alluded to, the firm did not attempt to get out of their contracts, although they involved considerable loss. Some American cases hold that there can be no recovery against lunatics in cases of this kind. In certain English cases, which will be found in the ordinary text-books, the opposite has been held. When it can be shown that an individual took advantage of an insane person to make a contract in his own favor, the courts would decide against the contract.

"A person who had been declared to be insane by an inquisition or commission could not, I suppose, be held responsible in a court for any contract; but in other cases it would be largely a question of argument before a jury, and of special decision by the judge.

"Let us now briefly consider the question of marriage contracts, in the case of the insane. The law recognizes insanity as an impediment to marriage. The insane person is in a condition similar to that of an infant under the law, that is, one who has not reached the age of 21 years. A marriage contract with a lunatic whose insanity has been legally or generally recognized would not be held to be valid, yet various cases arise in which marriage contracts with the insane are considered valid.

The "Rhineland case" was of some interest in connection with the question of marriage contracts, as well as in other respects. Mr. Rhineland is a member of one of the wealthiest families of New York, and is alleged to be insane. From the accounts which I have read in the newspapers, and in one or two

medical journals devoted to psychiatry, there would seem to be little doubt that he is in a delusional state. He is a shrewd and, superficially, an able man. I say 'superficially,' because the appearance of ability in these cases is usually only on the surface. He shot a New York lawyer who was the solicitor of his father's family, and was the instrument through which money was paid to him, on the supposition or delusion that the lawyer was having improper relations with his wife. He was declared sane. The case was four days before the Surrogate, and after testimony was given by experts on both sides, and evidence as to the man's past history was offered, a majority of the commission decided in favor of his sanity and a minority against it. The Surrogate decided in favor of his sanity, and under the law he is recognized as sane. In his case certain questions in regard to marriage arose. He had married a woman supposed to be in an inferior position in life, a domestic, I believe. Efforts were made to have them separated and to invalidate the marriage contract, under the idea that it was a marriage with a lunatic. These efforts not only failed, but the proceedings to find him a lunatic also failed. While a marriage with a lunatic is unlawful, the question of insanity is not always decided in accordance with the facts by judge and jury."

Dr. Mills then goes on to speak of the importance of physicians making careful examination of the mental as well as of the physical condition of patients under their charge who are likely to die soon, and wisely urges that physicians who are present at the time of making or witnessing wills should fully assure themselves as to the mental status of the testator, especially if the individual is possessed of considerable property.

"Not every man who is a lunatic (even under the law) is deprived of the right to make a will. A lunatic who has been certified to be insane, who has been declared by special legal process to be insane, and who has been an inmate of a lunatic asylum, has made a will disposing of his property, and this will has been upheld by the court. That will show you at once that the mere fact of alleged lunacy or the existence of real lunacy will not in every instance enable a contest against a will to be successfully sustained.

"Relatives are often unjustly deprived of their rights as a result of the peculiar mental condition of the individual at the time of making a will; and, on the other hand, those competent to dispose of their property are unjustly alleged to have been insane.

"The law is about this, that a person is regarded by the law as of a 'sane and disposing mind if he knows the nature of the act he is performing and is fully aware of its consequences.' These words are quoted from the law as found in text-books. This constitutes the phraseological rock upon which these questions are argued, but even this is capable of much discussion. A man may be aware of the nature of the act he is performing, and be aware of its consequences, yet he may be insane and do a very unjust act; but it is difficult to get a general principle to cover these important cases. It is like the old question of the plea of insanity

in criminal cases, many arguing that we should make the knowledge of right and wrong on the part of the alleged lunatic the point about which the question of his responsibility should hinge. One thing must be borne in mind, that bodily disease, no matter what it may be, will not, under the law, incapacitate a man from making a will, unless it can be proved that the bodily disease has so affected his mind as to render him incapable of judging, or, in other words, has made him not of a disposing mind. Various special phases of this question are of great interest. The question of delusions in insanity with reference to wills is one of these. It has been affirmed by courts that the mere fact of the existence of a delusion, or the fact that the person was a delusional monomaniac, would not invalidate a will. It practically comes to this: that in order to gain a case in a contest of this kind, it must be proved not only that the person was a delusional monomaniac, but that the delusion was of such a character as to interfere with his just judgment in making a will. In other words, a man may have hallucinations of hearing, or sight, or persecutory delusions of a marked character, and yet he may be able to conduct his business in a proper manner. Evidence could be brought in such a case to show that the man had been a delusional lunatic with hallucinations of hearing and sight for, perhaps, twenty years; and testimony equally strong might be given to show that during those twenty years he had not made a bad business contract. In such a case the will would probably hold.

"Mere eccentricities and peculiarities of wills do not invalidate them. Thus recently a maiden lady, fond of cats, left a large portion of her fortune for the establishment of an institution for the care and comfort of cats, and I do not know that the validity of this will has been questioned. A well-known gentleman holding certain peculiar views with reference to spiritualistic phenomena left a sum of money to this university for the investigation of this subject. It might be held that owing to these peculiar views the man was not in a mental condition to make a will, but the courts would probably not sustain such a contest. Another case is that of a man who believed in the old doctrine of the transmigration of souls. He left a will which held, certain parts of which were supposed to be connected with this peculiar idea. Simple eccentricities or peculiar religious or other views are not sufficient to invalidate the testamentary capacity. Still, in not a few of such cases injustice has been done to somebody. An institution may get the money which should go to a deserving family.

"One way in which the will of a lunatic is sometimes considered valid is on the old doctrine of lucid intervals. I was once asked in a lunacy trial by a dignified jurymen, 'Doctor, what is the difference between lunacy and insanity?' Practically, we do not recognize any distinction between the terms lunacy and insanity nowadays, but there is an old distinction. A lunatic in the old legal sense of the term was one who was insane, but had lucid intervals supposed to depend upon the changes of the moon. This



question of lucid intervals is one which is often brought up in will cases, it being claimed that although the individual was insane, yet he had a lucid interval. A man in this city had an attack of apoplexy in the morning. He was seen every three or four hours by different medical men during the twenty-four hours before he died. It was claimed that during this time he had made a will. As far as the medical witnesses had seen, he had been comatose much of this time. It was said that he roused up and made a will, but the will was not signed. It was said that he did not sign the will because of the paralyzed condition of the hand. This, however, is an extreme case. In many cases, as in circular insanity, where you have melancholia, then a lucid or sub-lucid interval, and then mania, etc., a will made between the attacks would probably hold in law. This old question of lucid intervals is an interesting one for discussion, and it is one on which contests are often based or resisted.

"Senile dementia is another condition on which a contest of a will is often based. Some most interesting cases are of this kind. One recent case is that of a man in West Virginia, in which a number of those connected with the University have given testimony. This man conveyed his property to certain of his relatives before his death, and thus deprived some of his children of their inheritance. It is shown by a hypothetical question, which has been answered by physicians, that he was not in a state of mind to decide justly, because of senile dementia. Senile dementia is something more than the normal deterioration of the aged. It is a peculiar disease with special symptoms."

CARLOS F. MACDONALD.

**Asylum Care of the Insane.** DR. B. D. EASTMAN, Superintendent of the State Insane Asylum at Topeka, Kansas, in his biennial report for the period ending June 30th, 1886, under the head of "General Management," says :

"It is a self-evident proposition that our eleemosynary institutions are the property of the people, are supported by the people, and are for the benefit of the people.

"It is our bounden duty to administer the trusts imposed upon us faithfully, honestly, and fearlessly, and no one feels more keenly its magnitude than he who has the responsibility. There must necessarily be one captain to a ship, one general to an army, and one head to an institution of this kind. The work is peculiar, and calls for knowledge and experience in widely differing directions. In the direct medical care of patients, there is need of medical skill and experience, while their control and management calls for quick insight as to character and psychological conditions. In the selection, training, directing, and disciplining of employes, there is need alike for intuitive feeling and judicial decision. In the planning and erecting of buildings, there is call

for mechanical and sanitary knowledge. In general administration, there must be vigilance and economy. In dealing with the public, there should be courtesy and sympathy, as well as firmness and decision. It is not to be assumed that any one can conduct the multifarious offices of such an institution and never make any mistakes. He who could do this would be more than human. But it may be conscientiously claimed that most earnest effort is made to accomplish the best results, and the officers and employes who have aided to this end are well deserving their meed of praise.

"To all who are interested in the welfare of this institution as citizens, as friends of patients, or as public officials, we say, 'Come and see us—see for yourselves what the State has done and what we are doing.' There is a great deal of unfounded suspicion and distrust of institutions for the insane, which will dissipate when knowledge is gained of their real workings. Ignorance and misrepresentation are at the bottom of most of the suspicions against State institutions for the insane, and sometimes even that which should redound to their credit is tortured into equivocal distrust. For instance, a statement circulated among the newspapers that a case had been found in Pennsylvania by the Board of State Charities which had been subject for a long time to the grossest abuse, was inferentially charged against the public institutions of that State, and made a text for suspicious inuendoes against insane asylums. The abuse of this unfortunate man had occurred 'in the house of his friends,' he having been for many years secluded with almost no care, in a very unsuitable room in his own house, whence he was removed by the State Board of Charities to one of the State institutions for the insane.

"One of our greatest anxieties in administration is the difficulty of securing suitable employes in many places, particularly in immediate care of patients. The welfare and comfort of inmates, and their recovery as well, depend largely upon the personal character of the attendants. In foreign asylums it is customary for persons to expect to make their life business the care of the insane, as attendants; here it is looked upon as a temporary make-shift, particularly by men. The duties of those directly engaged in the care of patients are very varied, and for their most successful performance require talent of no mean order. The attendant needs to control and direct those under his care, and at the same time to be a companion and an entertainer. He must also be an adept at household work, and fertile in resources. It often happens that he who entertains and pleases best fails in housekeeping, while the good housekeeper, who polishes the door knobs to the brightness of a mirror, rasps the patients' feelings with equal vigor. Hence the complaint is rife all over the country that attendants are continually changing, and so often unsatisfactory. But when the faithful, conscientious attendant is found, he is highly prized, and he may be assured he is laying up treasures, figuratively if not materially.

"In the interesting field of medical and moral treatment of insanity, we have tried remedies and agencies new and old,

sometimes with good results, sometimes without. Notwithstanding the fact that, in many cases, curative medical treatment is of prime importance, the moral treatment is often of greater usefulness. Removal from the worriment, the over-work, the unsanitary conditions, and the unsuitable food of many homes, relief from the distractions of business, replacing the morbid, nervous stimulations of distracted or frightened friends by the firm control of the asylum, occupying body and mind in new employments, cheering the drooping and melancholy and soothing the excited and irritable, are some of the elements of material and moral treatment of the greatest value, sometimes working rapid cures with but little medication. The providing of suitable bodily and mental exercise and occupation for our inmates is a difficult and delicate task, attended with many vexatious trials; and yet it is one of our best and most important aids to securing quiet, rest, and recovery. Suitable bodily exercise assists in the healthful performance of the organic functions, and the mind, occupied by agreeable activity, is not devouring itself. When overwhelmed by delusions or melancholy, or when violently disturbed by excitement, it is impossible to fix the attention upon avocations or amusements; but there frequently comes a time when the attention can be arrested and the mental powers turned to reconstructive rather than destructive tendencies. Sometimes, when prearranged efforts utterly fail, accidental opportunities engage the attention."

CARLOS F. MACDONALD.

#### THERAPEUTICS OF NERVOUS SYSTEM.

A noteworthy paper, and one which is probably destined to open up the way for further investigations, was read before the British Medical Association by Victor Horsley. The subject was the surgery of the brain with the reports of three cases on which the writer had operated.

The first part of the paper was devoted to a description of the technique of operations, treating in all detail the various steps to be followed, for which the reader is referred to the original paper in the *British Medical Journal*, Oct. 9th, 1886.

The three cases illustrative of the paper were as follows :

CASE I. was a young man, 22 years of age, who suffered from epilepsy as a result of an accident at age of 7, causing a depressed comminuted fracture of the skull, with loss of brain substance, at a point corresponding to the upper third of the ascending frontal convolution. The fits, which occurred in batches at time of operation, reaching three thousand in a fortnight, were almost always of the same character, usually commencing in right lower limb, and successively attacking right upper limb, right face, and neck. They were followed by right hemiplegia. The left side is not mentioned as being affected.

Operation performed May 25th, 1886. The bone around the old opening in the skull was removed, and a scar in the brain was

found, with other accompanying pathological conditions in the dura mater, etc. All the brain-scar tissue was removed. The operation was followed by complete paralysis of fingers of right hand, and by *incomplete loss of sense of touch* below the wrist, and *loss of muscular sense* in fingers.

Horsley, however, thinks it possible that the sensory disturbance may have been due to injury to some of the fibres coming from the gyrus fornicatus in the corona radiata. The motor and sensory paralysis disappeared in the course of the next two months. Up to the present time the patient has had no fits.

CASE II. was one of epilepsy in which the convulsions began with chronic spasmodic opposition of the thumb and forefinger (left), the wrist next, then the elbows and shoulders were flexed clonically, then the face twitched and the patient lost consciousness. The left leg, right leg, and right arm were then successively convulsed. Paralysis of left leg frequently followed a fit.

Dr. Hughlings Jackson diagnosed an irritative lesion, situated at the junction of the lower and middle thirds of the ascending frontal and parietal convolutions.

On operating, a cortical tumor was found in the suspected region, and removed. "Before closing the wound, the centre of the thumb area was removed by free incision. This detail Drs. Jackson and Horsley had resolved to carry out in the possible event of there being no obvious grave organic disease in order to prevent, as far as possible, recurrence of the epilepsy."

The operation was followed by partial motor paralysis of the left side of face, and complete paralysis of left arm and shoulder. Left incomplete hemianæsthesia developed. Later, all these symptoms disappeared, and at time of report there only remained weakness of grasp of hand and the fine movements of fingers were hampered.

The third case was diagnosed as epileptiform convulsions, due to an irritative lesion situated in the posterior third of the superior frontal convolution following injury of the skull. As before, scar tissue was found exactly at the spot diagnosed, and removed. One week after the operation, paresis of the right arm supervened. This, Horsley thinks, was hysterical. It had practically disappeared at time of report.

In the discussion following the paper, Erichsen, Charcot, and Hughlings Jackson spoke eulogistically of Horslev's results.

MORTON PRINCE.

**Trephining in Epilepsy.**—Dr. A. Hughes Bennett and Mr. A. Pierce Gould report in the *British Medical Journal*, January 1st, 1887, an interesting case of epilepsy apparently cured by operation. It has also some bearing on the localization of the visual centre.

The patient, male, æt. 36, with a good family history, received a severe blow about six years ago on the right side of the head. He was rendered unconscious for several hours, but there was no paralysis of any kind. The scalp was cut, but no injury to the

bone could be detected. The wound healed, leaving a cicatrix, the centre of which was three and one-half inches from the longitudinal fissure in a line drawn vertically two and three-fourth inches behind the external auditory meatus. The man returned to his work; six weeks later, he had a convulsion, during which it was noticed the movement of the limbs was limited to the left side. After this the attacks became more frequent, so that just before the operation they averaged one each week.

The loss of consciousness was usually preceded by a bright-red flash of light, followed by what were apparently visual hallucinations and maniacal excitement. The patient was so violent that he was confined, at different times, in an insane asylum.

Trephining was decided upon in the hope that there might be a possible depressed fracture or some other local or removable injury to the underlying cortex.

The operation was performed, with strict antiseptic precautions, by Mr. Gould. A large trephine opening was made at the centre of the cicatrix, a joint overlying the angular gyrus, but nothing abnormal was found in either the bone, dura mater, or cortex. Exploratory incisions were made into the cerebral substance, but neither cyst, abscess, or new growth could be detected. The wound was closed; no complication ensued, and the patient made a good recovery.

Since the operation, a period of six months, the patient has been entirely free from attacks, and is now able, for the first time in six years, to attend to his former occupation.

The cicatrix on the scalp and the subsequent trephining were situated over the region corresponding to the angular gyrus. The authors believe that the sensation of light at the beginning of an attack might be explained by the initiating irritation of the cortical visual centre; and that the mania following the fit, which appeared to be accompanied by visual hallucinations, may have been associated with some functional disturbance of this convolution.

While admitting that sufficient time has not elapsed to insure certainty as to the permanence of the so far successful results of the operation, Dr. Bennett thinks it advisable to add this contribution to a subject which, at the present time, is so much occupying the attention of the profession.

SANDERSON.

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DR. W. R. GOWERS and Mr. Arthur E. Barker have published (*Brit. Med. Jour.*, December 11th, 1886) a case of abscess of the brain, treated successfully by trephining and drainage. The case is also of interest in other respects. The case began as an otitis media. There was later double optic neuritis, inequality of the pupils, and vomiting. The mastoid antrum was first opened, but there was no discharge of pus; but some extremely fetid matter was washed out. The symptoms increasing, it was determined to trephine for cerebral abscess and search for one in the temporo-

sphenoidal lobe. The point on the skull selected was one and one-fourth inches behind, and one and one-fourth inches above the external meatus. This point, it was shown by dissections on the cadaver, is situated directly over the posterior part of the middle temporo-sphenoidal convolution. On exposing the brain, nothing abnormal was noticed. An aspirator needle (size of No. 4 catheter) was accordingly thrust into the convolution inwards, downwards, and forward; at a distance of about half an inch from surface a pus cavity was struck; nearly an ounce of intensely fetid pus was withdrawn. The lobe in the brain was enlarged by scraping away the cortex with a Volckmann's spoon, and a silver drainage tube inserted. The cavity was afterwards frequently washed out. Recovery followed. No mention is made of any mental impairment following the operation.

Dr. Barker makes some practical comments upon the operation. He adds that he thinks this the first case of abscess due to tympanic suppuration which has been correctly diagnosed, localized, and evacuated by operation with success. In this case the risks of the operation were enormously enhanced by the intense foulness of the whole suppurative process.

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**Hyoscine Hydrobromate as an Hypnotic in Private Practice.**—Drs. Francis L. and John R. Haynes contribute to the *Therapeutic Gazette* (September 15th, 1886) the results of their carefully recorded observations. The paper is based on the administration of 338 doses of hyoscine to 57 persons.

Notes are given of all the cases, which are divided into three classes.

Those in which hyoscine caused, I., sleep; II., delirium; and III., neither sleep nor delirium, but either no effect or various symptoms. The first class, 15 cases; the second, 13; and the third, 29.

The authors say, in some instances, little or no effect was noticed after hyoscine, but generally one or more of the following symptoms occurred.

1. Delirium, rambling, or muttering, with hallucinations of vision resulting in attempts to grasp imaginary objects.
2. Sleep, sometimes apparently natural, sometimes disturbed.
3. Intense reddening of the whole face, with sensations of heat affecting the whole body. It was not determined whether there was an actual elevation of temperature in such cases as in belladonna poisoning.
4. Muscular weakness, sometimes extreme.
5. Headache.
6. Dryness of throat and mouth.
7. Dilatation of pupil, with blurred vision.

The most remarkable variation was noted in the symptoms in different individuals, and sometimes in the same individual. Thus equal doses were given to two women under very similar

circumstances : one was poisoned ; the other fell into a pleasant sleep. In one case  $\frac{1}{100}$  gr. was followed by sleep, while gr.  $\frac{1}{100}$  produced delirium. In another, gr.  $\frac{1}{100}$  produced delirium ; and a larger dose,  $\frac{1}{80}$  gr., was followed by sleep.

The authors consider hyoscine extremely unreliable as a hypnotic, and that it should not be used in general practice, except in cases in which other hypnotics have failed. M. PRINCE.

**The Ætiology and Treatment of Migraine.** By PROF. A. EULENBURG. Reprint from *Wiener Med. Presse*, 1887.

The author discusses the various theories that have been propounded of late years regarding the ætiology of migraine; he is not thoroughly pleased with any one explanation, least of all with the *reflex* theories. He grants that there may be a kinship between epilepsy and migraine, but granting this, the gain is not a great one until we know more about epilepsy. The author is of the opinion that it will be best to regard hemicrania as a vaso-motor affection of the sympathetic nervous system, and to this we believe the majority of authors will at present agree. The remarks on the ætiology of the single attack are of greater interest. The attack is brought on by sudden variations in the endocranial blood-supply, the dilatation or contraction of the blood-vessels of the cerebral membranes producing irritation of the trigeminal filaments in the pia and dura mater. Those individuals would therefore be most likely to suffer from migraine who, by reason of some congenital defect, were either subject to sudden changes in the cerebral blood-supply, or had an unusually irritable trigeminal nerve, or were subject to both conditions. To prove this theory, Eulenburg has hit upon the ingenious theory of measuring the resistance of the two sides of the head, and he finds that, as a rule, the resistance is greater on the side of the migraine, particularly in the spastic form of migraine. Diminution of blood-supply accounts for increased resistance. (All this is in such striking accord with the theory here laid down that we are curious to know whether, and hope that, these facts and conclusions will receive further substantiation. Electrical resistance is chiefly a matter of the skin; the question arises : Does pallor of the skin imply anæmia of the membranes; redness of the skin, hyperæmia of the membranes ?)

The remarks on treatment have reference to recent suggestions. Massage—tapotement, effleurage, and more specially the use of the percuteur—is viewed with favor for the cure of a single attack. In the way of constitutional treatment, Eulenburg advises following out the principles laid down by Oertel, more specially in cases in which there is some marked circulatory disturbance. The writer also urges the use of salicylate of soda and antipyrin, the latter in fifteen-grain doses, to be repeated after an hour in the initial stage of an attack. (The present writer has tried antipyrin in a few cases of migraine and in typical trigeminal neuralgia, but for the

present he prefers caffeine, cann. ind. and iron, and ergot or nitrite of amyl, according to the nature of the attack.) Prof. Eulenburg has a good word to say also for static electricity, and mentions one case in which the application of the negative head-douche cut short an attack of hemicrania.

B. S.

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**The Value of Indian Hemp in the Treatment of a Certain Type of Headache.** By STEPHEN MACKENZIE, M.D. (*British Medical Journal*, January 15th, 1887.)

The headache described by the writer of this article is a very common one, but at times very difficult to treat. It is usually of a dull, continuous or subcontinuous character, attended sometimes with paroxysmal exacerbations. What is especially characteristic of it is its constancy. The headache may in some cases become aggravated as the day advances, but sometimes the opposite condition obtains, and it is worse at the early part of the day. Its situation varies; it may be frontal, temporal, or occipital. Usually, however, it is diffused. There is, as a rule, no local soreness or tenderness.

Nausea may be present; vomiting is usually absent. As a rule, there are none of the ocular phenomena characteristic of migraine, and the headache is not often hemicranial. Constipation is present in a certain number of cases, but removal of the constipation does not cure the headache. In some cases it is associated with disorders of digestion, but the same remark applies to these as to constipation. Headaches of this type may last for weeks, months, or even years. They are most common in young adults, and in persons in the middle period of life.

The nature of these headaches is obscure. They are not due to peripheral irritation or anæmia, but to some dyscrasia or diathesis.

It is in relief of these continuous or chronic headaches that Indian hemp is of the greatest service. The best results are obtained from the use of the extract. One-third of a grain, in pill form, is given night and morning for a week. If no improvement results, the drug can be gradually increased until two grains at night and one and a half grains in the morning are reached.

The important points in the treatment are the gradually increasing doses and the steady perseverance in the use of the drug.

The length of time over which treatment extends varies in different cases, usually several weeks, but rebellious cases require several months. As the malady recedes, the dose should be reduced, and it is advisable to continue the administration of the remedy for a week or two after the headache has disappeared.

SANDERSON.



## Book Reviews and Bibliographical Notes.

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**The Functions of the Brain.** By DAVID FERRIER, M.D., LL.D., F.R.S. SECOND EDITION. G. P. Putnam's Sons, 1886.

As a rule, second editions of a popular scientific work do not call for extended notice, nor for another critical review. Not so with Ferrier's "Functions of the Brain." This second edition is practically a new treatise, and bears splendid testimony to the immense additions that have been made to our knowledge of the anatomy and physiology of the nervous system (and of the brain in particular) during the decade that elapsed between the appearance of the first and second editions of this work.

That Ferrier has been an earnest student of the literature of the special subject which he is investigating no one can deny—at least no one who casts more than a cursory glance over these pages. He has assimilated a great deal of knowledge for which he is indebted to the investigations of others, and yet on the great question of the localization of cerebral functions—by which this work must be judged as the first edition was—he stands as he formerly did. "The principal doctrines formerly advocated . . . are maintained in all essentials unchanged." What does this signify? Does this mean that Ferrier's experiments and conclusions are so far beyond reproach that the position he assumes is an impregnable one; or must we regard him as an enthusiast who can neither appreciate the good in the methods of others nor the blemishes in his own? Let us see!

Before the first edition of Ferrier's book appeared (in 1876), the question of the localization of functions has been studied by Flourens, Brown-Séquard, Schiff, Fritsch and Hitzig, and Nothnagel, while Goltz had published his first article only a short time previously; since that time the question has been studied and discussed by Munk, Goltz, Exner, Loeb, Luciani and Sepilli, Ferrier, and possibly a host of others. Experimental physiology and human pathology have given strong evidence in favor of a specialization of cortical centres: Goltz alone, of late years (supported, to be sure, on various occasions by no lesser lights than v. Gudden and Nothnagel), has stood out in open opposition to the doctrine of a strict localization, and has brought upon himself the criticisms and often the vituperations of the "localizers." To the credit of Ferrier be it said that he treats Goltz's conclusions with the con-

sideration which a true genius<sup>1</sup> deserves. But there has been little harmony among the "localizers" themselves. Fritsch and Hitzig could not agree to the exact localization of Ferrier's centres. Munk also ridiculed Ferrier's centres, and, by way of return, Ferrier snubs Munk. Loeb, under the inspiration of Goltz, demolishes Munk's visual centres, while Luciani and Sepilli agree in some points with Munk, in others with Goltz or Exner, and in a few with Ferrier. Where lies the truth? In all likelihood, midway between the extremes.

In spite of all the hostile criticism that has been passed upon it, Ferrier still upholds his old method of experimentation—the method of determining the locality of the various centres in the cortex by *electrical stimulation*. The one serious objection to this method is, that there is no telling where the current will diffuse to, and that movements resulting from electrical irritation of the cortex may be due to the influence of the currents, not upon the cortical cells, but upon the subjacent white fibres. But Ferrier argues (p. 230), "if the medullary fibres are differentiated in function, the regions to which they are distributed must be similarly differentiated." True; but, with areas as small as those of the author, I can hardly conceive that fibres going to *neighboring* areas should not receive part of the current as well. (Some of the author's recent and most brilliant experiments have been based upon the method of removal with the knife, and in this the application of antiseptic principles has stood him in good stead.) All the old facts and most of the old cuts fixing the little fanciful circles upon the brains of jackals, monkeys, dogs, cats, and rabbits appear again; but as the method is not convincing, the conclusions cannot be credited. It will be seen that, with very few exceptions, the circles representing movements of distinct muscular groups remain within the areas now agreed upon by pathologists at least, as representing the leg, arm, and face centres respectively. In other words, Ferrier attempts a minute differentiation which no one else is willing to adopt. And certain it is that any one who, like the present writer, has seen Goltz remove a half-dozen or more of Ferrier's centres at a single operation,<sup>2</sup> and who afterward observed the same animal running about as well as ever, will not believe in these carefully circumscribed areas, whatever other view pathological findings may induce him to hold. We have dwelt at some length upon this question of circumscribed motor areas to insist

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<sup>1</sup> The fact is often overlooked that Goltz has proved himself the keenest of all observers; but for his ingenious suggestions (to mention a single fact only), the methods of testing the disturbed sensory and motor faculties of animals would be far less refined than they are at present.

<sup>2</sup> The unfortunate experiences of Goltz in exhibiting (at London and Berlin) the brains of the animals operated upon renders it doubtful whether he has in every case removed an entire occipital cortex, or an entire arm centre, and the like; but that many of Ferrier's centres have been removed without the expected result cannot be doubted.

upon the points of difference between Ferrier's views and those of other investigators.

In determining the location of the sensory centres, the author very naturally makes liberal use of the facts of human pathology. His view of the value of pathological findings is the orthodox view of a physiologist: "Clinical cases are mainly valuable as confirmatory of physiological experiments, and more especially as supplying negative instances. A case, however otherwise complicated, of total destruction of a region in which a certain function is supposed to be localized, without loss or impairment of the function assigned to it, outweighs a thousand positive instances in which a causal relationship seems to be established between the particular region and the function in question." With this most of us will be disposed to agree in the main; but, after all, too much importance must not be attached to *single* cases. Kussmaul has recently reported two cases of total loss of first temporal convolution without sensory aphasia, and yet, unless negative instances are multiplied, there is good reason for associating sensory aphasia with a lesion of the convolution in question. In the location of the various sensory centres Ferrier is again at variance with almost all other authors.

In the first edition, Ferrier relegated the visual centre to the angular gyrus, electrical excitation of which produced movement of the eyeballs, contraction of the pupils, etc., which the author interpreted to be reflex movements consequent on the excitation of subjective visual sensation (p. 164 of first edition). At present, no one would argue that visual impressions must be received (apperceived) in the same area from which movements of the eyes are excited. Association fibres would come into play here.

In the second edition, p. 271, the author says: "The visual centres embrace not only the angular gyri, but also the occipital lobes, which together I term the occipito-angular regions." In the chapter that follows (*mirabile dictu*) the author takes all possible pain to show that the occipital lobes have little, if anything, to do with vision, and what the exact function of the occipital lobes is, according to Dr. Ferrier, we challenge any one to find out. If, angular gyrus = 1, and occipital lobes = 0,  $1 + 0 = 1$ . Why not retain the angular gyrus as sole visual centre?

The relation of the angular gyrus to vision is explained by other authors on the ground that the optic radiations into the occipital lobe pass beneath the angular gyrus, and that lesion of the angular gyrus could interfere with these optic fibres. In one case, which was examined with regard to this point, Ferrier states (p. 282): angular gyri had been completely obliterated, but the occipital lobes and the optic radiations . . . passing backwards into them were uninjured. Was this determined by microscopic examination or by mere superficial inspection? No further statements are made, and the proof of Ferrier's position is, therefore, extremely unsatisfactory.

With the exception of Ferrier, clinicians and physiologists believe that the occipital lobes have much more to do with vision and with the special disturbance known as hemianopsia than the angular gyrus has. Ferrier claims (p. 295) clinical facts . . . do not establish any relation between hemiopia and lesion of the occipital lobe as such, apart from the angular gyrus. It is very strange, indeed, that Dr. Ferrier does not take note of the conclusions of two American authors who have proved that such a relation does exist. The cases analyzed by Dr. Starr and Dr. Seguin have established this relation, and both these articles must surely have been in the author's hands before writing his preface.

Dr. Seguin's facts showing the relations of the cuneus to hemianopsia has met with general favor, and since the publication of his article, further cases in corroboration of his views have been reported by Seguin, Gruening, and Hun. When Meynert stated ("Psychiatry," p. 144) that Ferrier's centres have met with opposition from *all* other authors, British critics regretted Meynert's "national bias." We beg to state that in making the above strictures, we are not actuated by any such bias, but that we have not come across a more remarkable exhibition of "*hedging*" than is offered in this chapter on the visual centre.

Of the other centres, we have only to say that Ferrier locates the function of hearing in the superior temporo-sphenoidal convolution (in general agreement with other authors), tactile and general sensibility is referred to the hippocampal region; that the hippocampal lobule has relations to the sense of smell, while the author does not venture to indicate where taste has its central seat. How about tactile centres near the motor areas?

We do not feel that the question of cortical localization has been cleared up by the publication of this second edition. Coming as it does after the excellent monograph of Luciani and Sepilli, it marks a retrogression rather than an advance toward more perfect knowledge. Of this discussion on cortical centres, Ferrier may well say *magna pars fui*, to which we add, *non maxima*. Aside from other considerations, we do not think this book a safe one to put into the hands of students of physiology or psychology, unless it be to make them acquainted with one extreme view. The contest is still waging over this question of localization, but when such rational localizers as Luciani and Sepilli state that disturbances of vision can be caused by lesion in so many different parts of the brain that, properly speaking, the visual centres cannot be accurately limited, we leave Ferrier, Hitzig, and Munk, and incline distinctly toward the views of Goltz. We have no intention of entering any special plea at present, but we have a strong conviction that the conservative opinions on localization will in the end carry the day. However specialized the functions of different parts of the cortex may be, he who analyzes the simplest act—plucking a flower—will note how widely separated portions of the cortex will be engaged in the performance of this single act. But to return to the work: a great deal of excellent information has been col-

lected and sifted regarding the anatomy and physiology of the spinal cord, cerebellum, and cerebral ganglia. All these chapters are excellently written, and the subject matter is discussed in an entirely impersonal way. We commend them as readable summaries of our present knowledge. The typographical work is beyond praise.

B. S.

## Society Proceedings.

### NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, December 7th, 1886.*

C. L. DANA, M.D., *President in the Chair.*

DR. W. R. BIRDSALL presented a case of progressive muscular atrophy with bulbar symptoms.<sup>1</sup>

DR. E. C. SEGUIN had seen very few examples of unilateral progressive muscular atrophy. He had at present one patient under observation in whom the muscular atrophy was limited to one side, presenting the electrical and other characters of progressive muscular atrophy.

DR. W. M. LESZYNSKY had a girl, aged seventeen, under observation in whom the atrophy was unilateral, affecting only the supraspinatus, deltoid, and a portion of the trapezius.

#### SELF-ABUSE IN ITS RELATION TO INSANITY.

DR. E. C. SPITZKA, the author of the paper, after citing the views of the classical writers, stated that the question of the existence of a special form of insanity, due to self-abuse and to nothing else, was complicated by the existence of another well-demarcated affection known as the insanity of pubescence. The mental diseases due to self-abuse usually occurred at the same period of life as the latter disorder. This fact explained the similarity of many clinical features between them. The question was further complicated by the fact that hebephreniacs (sufferers from pubescent insanity) are often addicted to self-abuse, and that thus the features of one disorder may be engrafted upon the other.

The continental authorities do not recognize a special form of masturbational insanity in their tables. Schüle, it is true, speaks of *onanistic insanity* in the sense in which Maudsley uses that term; but he assigns no part to it in his classification, and dis-

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<sup>1</sup> To appear in the next number of this JOURNAL.

poses of it in a few lines. Krafft-Ebing recognizes the vice as an etiological factor, and speaks of such and such forms of insanity on a masturbational basis. He, as well as Schüle, with the majority of recent German writers, follows Ellinger in attributing to the *masturbatory neurosis* a relation to the development of insanity analogous to hereditary and other admitted predisposing and determining factors. I have yet to find any dissent expressed by these authorities from the position taken by Emminghaus, who claims that, owing to its causal relationship to widely differing forms of insanity, it is not proper to speak, as Skae does, of a special form due to masturbation. This critical remark would seem to be supported not only by the clinical facts accessible to every observer, but also by the confusion existing among those writers who have attempted to define and demarcate such an affection. Skae speaks of a peculiar imbecility and shy habits as characterizing the disorder among the youthful, and suspicion and fear and scared looks, palpitation and feeble bodies as found in older victims, who gradually pass into dementia. The most distinguished follower of Skae attributes the following symptoms to that form of insanity of which masturbation is the chief cause and "the chief symptom present," giving "the whole case distinct features"; exaggerated self-feeling, conceited shallow introspection, frothy emotional religious notions, and a restless unsettled state with foolish hatchings of philanthropic schemes. Luther Bell, who with Isaac Ray was among the earliest to attribute special symptoms to insanity caused by masturbation, furnishes a very faithful picture of certain cases, whose particular feature he describes as being a tendency to dementia, a loss of self-respect, a sulky, mischievous, and dangerous disposition, and a subjectively irritable and depressed state of mind. Griesinger, who does not recognize a special form and denies specific characters, admits that the majority of cases are marked by a profound dulness of sentiment and mental exhaustion, by religious delusions and hallucinations of hearing, and a rapid transition to dementia, in the event of incurability, which latter is the usual issue.

The effect of masturbation on the mind and nervous system varies according to the age at which it is commenced. Like other agents which are injurious to the developing brain, such as epilepsy, alcohol, and syphilis, its effect is most rapid and serious in younger children, less so in adolescents, and least marked in adults—unless protracted. For very young infants it causes a

profound deterioration, manifesting itself in convulsive, choreic disorder, and imbecility. In those who masturbate between the fifth and tenth years the effects seem to be manifested chiefly in arrested brain nutrition. Spontaneity of thought and action is absent with such children; they do not play as their comrades do.

There are a number of other circumstances which modify the development of mental disturbance in masturbators. The age between twenty and thirty-five is pre-eminently the period of somatic introspection. It is at this period, if at any, that the average man begins to think about his bodily condition. In these years men weigh themselves, discover that they have too much or too little flesh, develop slight gastric or intestinal disorders, reflex nervous symptoms, or indulge to excess in tobacco, in baccho, and in venere, and consequently are on the *qui vive* for the occurrence of cardiac, renal, or venereal disease, or of sexual disability. It is at this period that the results of masturbation are most deeply felt by a large proportion of the victims of that habit. The prevalent tendency of his age and of his associates of the same age carries him into a veritable nosomania. Perhaps, also, he attempts, under lay or medical advice, to accomplish coitus, and fails. It is for this reason that we find the larger portion of cases of insanity due to masturbation developing between the twenty-fifth and thirty-fifth year classified as "hypochondriacal paranoia."

A number of typical histories were then related, from which the author drew the following conclusions: 1. Self-abuse is an etiological factor in a large number of cases of insanity but only those cases should be designated as insanity of masturbation in which the connection between the excesses and the symptoms is direct; 2. Self-abuse, to produce insanity, must have been carried very far or the subject must be predisposed. Often onanism can be traced in other members of the family, and very often it is found that the maternal ancestry is a weak one; 3. Mania, melancholia, and epilepsy occasionally occur in young masturbators, the former two usually having a favorable prognosis; 4. Stuporous insanity and katatonia are both common, and the former presents good prospects; 5. The forms thus far mentioned when occurring in masturbators present no essential difference from the typical psychoses. They should therefore be designated as mania, melancholia stupor, etc., *from* masturbation, and not as masturbational insanity; 6. There is a chronical delusional insanity in grown persons who have been devotees of self-abuse, and it is usually a hy-



pochondriacal *paranoia*. Clinically, it is very like typical *paranoia*, and etiologically it is not the direct result of self-abuse, but rather of an intermediate neurosis, a cerebro-spinal irritation which is due to self-abuse; 7. Finally, there is a form of insanity developing about or after the period of puberty which does merit the name 'masturbational insanity'; it is chronic, has a tendency to agitated dementia, is characterized in its early period by anxiety, timidity, suspicion, fear, and a cowardly, mean disposition. Later there are confusion, meddlesome, aggressive behavior, vague delusions, loss of memory, and deterioration. After these are observed spells of fury or destructiveness. This form is never due to any other cause, and resembles no other form of insanity than the one already alluded to; 8. It is not always possible to differentiate between the insanity of pubescence and the form described. But where the former disorder is uncomplicated by the latter, it may be known by a history of peculiarities in infancy and childhood, by the greater constancy of the mental state which in onanists is exceedingly variable. Hebephreniacs are more apt to be expansive in their notions, more inclined to favor projects of a chimerical character; in other words, insanity of pubescence is the *paranoia* of adolescence, and masturbational insanity the pre-senile dementia of the same period of life.

DR. RALPH L. PARSONS made some remarks with reference to the treatment. The diet should be principally vegetables and milk, with little meat and stimulating condiments. As the patient sought solitude, he should be thrown as much as possible with others, not alone of his own sex, but also of the opposite sex. He should be kept occupied, and manual labor of some form, like farming, was best. He knew of no special benefit to be derived from medicinal treatment, as with the bromides, or with the application of irritating substances to the penis. Cutting off the prepuce might be of advantage in some cases. The patient should be closely watched day and night; mechanical appliances might sometimes be necessary; moral influence could be depended upon to a certain extent.

DR. KELLOGG agreed with the author in the conclusions arrived at in the main. But he would like to know Dr. Spitzka's views as to the relative importance of artificial sexual indulgence and indulgence in the natural manner as factors in the production of insanity. Masturbation was a wide term, and ought to be defined. The effects in some cases were more observable in spinal lesions,

in others in cerebral lesions. He believed that masturbation itself was not capable of producing insanity in a person of sound heritage. He was convinced that it was capable of suspending mental growth and producing forms of imbecility in those of sound parentage. He knew it could produce insanity at the time of pubescence, and there were persons of mature age who had a predisposition to insanity in whom the attack was excited directly by sexual excess. Occasionally persons indulged to excess for a year or two only, as did sailors sometimes when on long voyages. Masturbation was also capable of producing insanity in old persons who were on the decline; it hastened dementia. He did not think there was a peculiar set of symptoms; the age of the patient, his education, his heritage, his whole mental make-up influenced the symptoms more than the exciting cause. He did not believe it possible to separate masturbation from other forms of sexual excess, and the title "sexual abuse" would have been more appropriate, because more comprehensive, than "self-abuse."

DR. NOYES said that of the cases referred to by the author as having been cured he had seen one in the Bloomingdale Asylum and he attributed recovery in that case to transferring the patient to a farm, where his whole mode of life, including diet, was changed and for the better.

DR. L. C. GRAY thought the author had given an accurate description of the mental disturbances often seen associated with the habit of masturbation, but he asked if he did not also find similar mental disturbances in individuals who were not masturbators.

DR. SPITZKA replied that in individual cases he had, but not in groups of cases as occurred in masturbators.

DR. GRAY had seen the mental disturbances described in patients addicted to masturbation, but he had been unable to decide as to what extent masturbation could be considered as a cause or simply an associated habit. He had two cases in mind in which that group of symptoms were followed in the course of a few weeks by masturbation in individuals who had not previously been addicted to self-abuse. He had seen the same symptoms follow excessive sexual intercourse. He had in some cases noticed very exaggerated and extensive cremaster reflex.

In closing the discussion, DR. SPITZKA said that there were undoubtedly some forms of sexual vice which were physically as injurious as onanism; but he had not seen a sufficient number of cases to enable him to say anything about their mental sequelæ,

unless he cared to risk being premature. He had known epilepsy and stupor to follow natural sexual excess in a young person, and parietic dementia in more than one cunnilinguist and sodomist. The form he had sketched was, as far as his experience went, only found in masturbators. While he admitted, with Dr. Kellogg, that the single act of onanism was physically not a formidable thing, not much, if anything, different from normal coitus, there were two respects in which the onanist and libertine differed most widely, one was a moral, the other a physical feature. The onanist practises a secret crime, the social and gregarious element is excluded. Knowing that his act is despised, he becomes inclined to suspicion and fear of discovery. A libertine cannot exceed beyond a certain limit. Coitus requires a certain condition of the organs, which implies the existence of certain normal energies; when these fail, the limit is set to further excess. With the onanist it is very different. There are masturbators who require no erection; yea, who succeed in their injurious act without any manipulation. The consequence is that they pass far beyond the limit set by nature to natural excess, and no calculation can be made of the damage done.

Dr. Parson's dietary propositions were indorsed by the highest authority. Individually, the speaker was not decided in his own mind whether a highly nutritious diet would prove injurious in certain phases.

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*Stated Meeting, January 4th, 1887.*

C. L. DANA, M.D., *President, in the Chair.*

DR. W. H. PORTER presented the knee joint and spinal cord in a case of spinal arthropathy, and the spinal cord in a case of acute tabes dorsalis of six weeks' course. (To appear in the April number of this JOURNAL.)

DR. MARY P. JACOBI read a paper entitled,

NOTE ON APHASIA WITH REFERENCE TO LOSS OF NOUNS.<sup>1</sup>

PERIPHERAL NEURITIS AND THE PAINFUL PARALYSIS OF EARLY LIFE.

DR. H. D. CHAPIN read the paper, and said there had been great scarcity of autopsies in comparison to the frequency of paralysis in children. For that reason, a very careful clinical study was necessary, interpreted in the later knowledge of anatomy

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<sup>1</sup> See p. 94, this JOURNAL and volume.

and physiology of the central and peripheral nerves. The writer had met an atrophic form of paralysis differing at its inception, development, and result from the spinal paralyses with which he was familiar. Most of the autopsies had been made many years after the paralysis, when death had taken place from some other cause. There having been but few autopsies in proportion to the number and variety of cases of paralysis, there seemed rather a slender basis for the theory of exclusive spinal paralysis of childhood. Laborde's case is mentioned where a tabetic neuritis existed with sclerosis of the antero-lateral horns, while the ganglion cells were found normal. Robin's case was cited, where no lesion of the cord was discovered. Such a case would show that paralysis is not necessarily always spinal. Barwell's theory was incidentally mentioned. He claimed that infantile paralysis is purely peripheral, involving the ultimate fibrillæ of the nerves among the muscular elements. Later, Leyden advanced a more rational explanation when he considered that in atrophic paralysis there may be neuritis with spinal cord lesion, and that instead of such forms of paralysis always originating in the spinal cord, they may have their beginning in any part of the motor apparatus, then spreading to other parts, or remaining limited to the part first affected. Leyden claims that where complete recovery takes place the morbid processes always remain peripheric. Leyden's theory is accepted by the author of the paper as affording satisfactory explanation for certain cases that he has observed clinically. The histories of three cases were given in which paralysis was gradual at the onset, and attended by great and persistent pain. Pain was one of the most marked symptoms, and principally at the extremities—legs and feet. Most of these cases lasted several months, and then, to the surprise of the writer, slowly recovered. Atrophy was present. One of the cases appeared strongly rheumatic. In the other two, the cause was uncertain. Malarial poisoning appeared to be able to produce a more or less severe form of multiple neuritis resulting in paralysis. The history of a mild case was given which recovered under quinine.

Several cases were related in which children with malarial fever were seized with painful paralysis, lasting in one case over four months, followed by recovery. A possible explanation of pain is, that it is due to the marked general congestion of the gray matter of the spinal cord. The author stated that in his cases, and others like them, there were no other symptoms showing irritation

in the deeper parts of the cord. The general congestion or myelitis should cause bladder symptoms, bed sores, and other disturbances. Histories of two cases of lead paralysis in young children seen by the author were given. The lesions were regarded as largely peripheral. Cold usually attacks by preference the peripheral system of nerves. The loss of power sometimes following rheumatism is also probably of this nature. Any morbid blood condition appears able to produce a peripheral paralysis in early life, particularly the acute infectious diseases, especially diphtheria. The lesion in diphtheria is now known to be largely peripheral.

The object of the paper is suggestive, not dogmatic. The author has gladly availed himself of recent studies in peripheral neuritis tending to throw light upon some of the paralyses of children which have caused much perplexity. The great differences in the clinical behavior of paralyses in early life, in duration and curability, must admit of different anatomical and pathological explanations.

DR. M. ALLEN STARR did not know of any recorded case in a patient under twenty-four years of age in which a lesion of a peripheral nerve had been found at autopsy which would account for the paralysis, and while there was great probability in the statements made by the author, yet they lacked confirmation by autopsy. The points had been brought out very well with regard to the distinctions between peripheral neuritis and anterior poliomyelitis, but he would take exception to what had been said regarding the rapidity of the onset. There were many cases of acute onset of peripheral neuritis, the patient having fever and chill, and the limbs within twenty-four hours becoming entirely immovable. Too much attention could not be given to the usually great tenderness in the muscles and nerves in multiple neuritis.

DR. RUDISCH said that he was the first to observe, as long ago as eight or nine years, a form of polio-neuritis leading to paralysis and atrophy, and which followed affections of the joint, often so-called rheumatic affections. We now saw often enough an affection of one or more joints with inflammation, followed after awhile by paralysis, usually curable.

DR. FISHER said a girl 13 or 14 years of age was brought to the dispensary with the history that one morning after taking cold she woke up with paralysis of the upper and lower extremities.

At that time, she had only paralysis in one leg, which, he supposed, without thought of neuritis, to be ordinary polio-myelitis. Rapid improvement took place, and she recovered within six weeks, and the minister who brought her reported the case as one cured by faith. Dr. Fisher was unable to say whether the case was an affection of the anterior cornua or one of multiple neuritis.

DR. SACHS thought we should not be influenced too much by the present fashion, and call all or most of the cases formerly regarded as polio-myelitis cases of multiple neuritis. Referring to one of the cases related in the paper, and the symptoms of pain, he said too much stress should not be laid upon it unless it was severe, persistent, and located distinctly along the tract of a peripheral nerve.

DR. LESZYNSKY regarded pain as a prominent symptom of neuritis, absent in the majority of all of the cases of simple polio-myelitis. The pains present in some of Dr. Chapin's cases reminded him of the pain from straining of the tendons of the extensor muscles which had been for some time in a paralyzed state.

DR. CHAPIN thought that a neuritis would occasionally explain cases which could not be accounted for on the supposition of a spinal lesion, and he would rather say halt ! to the universal spinal cause of disease. With regard to pain it was difficult to locate it along a particular nerve in children, but in his cases it was acute and persistent, and not due simply to stretching of tendons.

## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, November 22d, 1886.*

*The Vice-President, CHARLES K. MILLS, M.D., in the Chair.*

DR. E. C. SEGUIN, of New York, read by invitation

### NOTES ON THREE CASES OF TROPICAL BERI-BERI, AND ON SOME SIMILAR AMERICAN CASES.

Having been courteously invited to participate in this evening's symposium on neuritis, I bring my mite of contribution in the shape of the relation of three cases of beri-beri, which, coming from tropical parts, have been under my care. That this disease is rarely seen in our latitudes, though very common in South and Central America and in the islands of the Gulf, I infer from the fact that it has not, to my knowledge, been discussed in medical societies or in private relations in New York during the past ten or fifteen years.

My apology for relating the following cases is that they well illustrate the prominence of neural symptoms in beri-beri, and that they justify the opinion expressed by a number of observers (Dr. B. Scheube and others) that beri-beri, or kak-ke, as the affection is called in Japan, is essentially a multiple, poly, or pan-neuritis, occurring as an endemic infectious (?) disease in many countries of the globe in both hemispheres. That beri-beri, or kak-ke, is nothing more than a multiple neuritis I am not prepared to say, and, indeed, my experience is so extremely limited that I can only use my cases to illustrate the probable neural origin of many of the symptoms of the disease.

In looking for notes of cases of multiple neuritis occurring in my local practice I found several. One, very typical, was recorded under the title of alcoholic polyneuritis; others were included in my essays on poliomyelitis anterior,<sup>1</sup> as examples of that disease.

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<sup>1</sup> "Myelitis of the Anterior Horns; or, Spinal Paralysis of the Adult and Child." New York, 1877. (Private edition, 1874.)

These three cases were marked by the ordinary symptoms of multiple neuritis: numbness, and partial anæsthesia, irregularly distributed atrophic paralysis, with degenerative reactions; and, besides, œdema of the extremities. I was struck by the resemblance of these cases, thus symptomatized, to mild cases of the mixed form of beri-beri. There is quite a resemblance, if not a similarity, between Cases I. and II. of the category of beri-beri and Case I. of the category of indigenous neuritis.

Another aspect in which a study of beri-beri is of interest to us is that the disease may take root and acquire considerable development in the United States. It appears that the affection has prevailed in Para, Brazil, for only a few years, perhaps only since 1878, having been brought there from Southern Brazil and Paraguay; and, as it is not uncommon in Cuba (at least the so-called wet form), it may very well show itself in the Gulf States.

With this brief preface, I pass on to the cases themselves.

*First Category: Case of Beri-beri.*

*Case I.*—A Cuban lawyer, aged twenty-six, consulted me September 9th, 1885, with letters from Drs. Landeta and Desvernines, of Havana, of which the following is an abstract: Married man of good constitution. Never had syphilis, scrofula, or paludism: never seriously ill. In summer and autumn of 1884, dyspeptic symptoms.

Symptoms of present illness: (1) Early in November (1884), pains in lower third of both legs. (2) In a few days these parts were slightly œdematous, while the malleoli and feet remained free from swelling. (3) Simultaneously, cardiac palpitations; rapid pulse, from 110 to 120. (4) Some muscular weakness was noticed in legs; walk difficult and done on tip-toe, or on forward part of foot. (5) Toward end of November the pulse was steadily 120 or more beats; large objective palpitations and souffles were noticed over cardiac area. (6) Edema appeared in the face, invaded the whole of the legs, thighs, feet, and genitals. (7) The muscles of the thighs and legs became soft and painful to touch; there seemed to be intramuscular œdema. Toward the end of the month patient was unable to leave the bed; the palpitation was intense, and there was great dyspnœa. The thenar and hypothenar eminences became soft; the patient could no longer write, or use fork, spoon, etc. Later the weakness became such that the legs could not be lifted from the bed: face was nearly twice its normal



size. The patellar reflex was abolished. Some muscles showed loss of contractility under electricity (*vide infra*).

As a summary of symptoms in the fourth and fifth weeks of the disease, it may be said that there were anasarca, paralysis with electrical changes in certain muscular groups, cardiac bruits, with pulse up to 140 beats per minute, dyspnœa. At no time was there fever, and no albumin or sugar was ever found in urine.

Under the use of infusion of digitalis, cinchona, and nutritious food, these symptoms abated, and there was decided improvement by the end of December (eighth week). Under electrical treatment and sulphur baths the motor paralysis gradually passed off, and the patient was able to stand in March: steady improvement thereafter continued, the cardiac symptoms had about ceased by the end of December.

Dr. Desvernine's letter refers more particularly to the state of the heart, and to the electrical reactions. At the height of the disease the heart was enlarged, more especially the left ventricle, and the murmur heard was a mitral regurgitant one. These signs were due, Dr. Desvernines believes, to myo-cardial lesions; the endocardium being unaffected. This view is borne out by the disappearance of the symptoms.

There was an apparent pseudo-hypertrophy of all muscles of the body (œdema). A piece of muscle removed by Duchenne's trocar showed simple atrophy of its muscular fibrillæ with intramuscular œdema and proliferation of the connective tissue. The electrical reactions were not taken during the acute period of the disease, but afterward were very fully studied, and there were found with both currents the "reactions of degeneration, characteristic of perverted nutrition in nerve and muscle."

The diagnosis made by Drs. Landeta and Desvernines was *beriberi* or *kak-ke*, of the mixed form, and Dr. Desvernines recognized in the case the grounds for the views advanced by Scheube and Balz (from studies of the disease in Japan), that it is essentially a multiple neuritis. It is interesting to add that, while the wet form (anæmic œdematous type) is well known to the physicians of Cuba and Havana, not one of several who saw this case with Dr. Landeta had seen an example of the mixed form.

When I examined Señor M. about ten months after the beginning of the attack, he presented no objective symptoms except a degree of abnormal flushing of the face. Excepting a slight quantitative reduction in contractility of the calf muscles, I noticed no

abnormality in the faradic and galvanic reactions of nerve, trunk, and muscles. The pulse was slow, from 57 to 63, and the heart normal in size and sound. The patient complained of occasional vertigo, and want of power of mental concentration. He was well October 10th.

*Case II.*—Mr. J. G., a married man, thirty-eight years of age, a native and resident of Panama, was brought to me, for consultation, by Dr. Pio Rengifo, of New York, on November 17th, 1885. Is a tall, strongly-built man, who has enjoyed good health, except that sixteen years ago he had a single chancre with suppurating buboes, but no secondary symptoms. Had a year's treatment with Dupuytren's pills and iodide of potassium. A healthy child has been the result of marriage. In the past eight years occasional palpitations, supposed to be due to gastric disorder. Is temperate in use of stimulants. About two months ago noticed numbness in toes and fingers. This has continued as a constant symptom, affecting tips of fingers, toes, and forward part of soles; on lying down, however, there has been a certain sense of numbness in the legs and forearms. Pains appeared later, of two kinds: first, aching pains in the calves, which were hard and tender; second, sharp pains in spots in fingers, legs, and feet; not, however, repeated pains in spots, with cutaneous hyperalgesia (not fulgurating pains). Knees were weak, and toes seemed cramped. Had sensation of band around toes. Had no vesical paresis, loss of sexual power, or spinal pains; no abnormal reflexes in legs, no diplopia. In last three weeks distinct œdema of feet and legs; has noticed that the calves measure two or three centimetres more in the evening than in the morning. During the past fortnight there has been marked improvement in power of legs, and in the paræsthesiæ.

*Examination.*—No objective symptoms about eyes or face. Grasp: right side, 38 and 32 degrees; left, 31 and 22 degrees; decidedly weak for a man of patient's muscular development and size. Sensibility is not positively diminished in fingers, but there is some confusion in estimating number of points applied; distinguishing texture of cloths well. No analgesia. Fingers are skilful; handwriting unchanged.

Lower extremities: no patellar reflex; walks a little awkwardly, with slapping of the feet; but, strictly speaking, there is no ataxia. Staggers when standing with eyes closed and feet together, but with eyes open stands fairly well, even on one foot. Sensi-

bility to æsthesiometer is dull, but he localizes impressions well; no analgesia. Those movements of toes which are controlled by interossei are limited. There is yet a trace of œdema over the tibiæ. No electrical examination.

I concurred in Dr. Rengifo's diagnosis of beri-beri or multiple neuritis, and the same opinion was expressed by Professor Charcot, who saw Mr. G. shortly after. I recently learned that entire recovery took place in three months. It may be of interest to add that Mr. G. presented complete transposition of the viscera, his heart (the sounds of which were normal) being distinctly on the right side of the sternum, and his liver on the left side; he was, however, right-handed originally.

*Case III.*—Señor J., aged fifty, a native and resident of Para, in Brazil, was referred to me by my friend, Dr. Charles E. Simmons, on November 11th, and is now under observation. Former health good; never had beri-beri or severe malarial fever. Subject to "bilious attacks." In August of present year had bleeding hemorrhoids for three weeks, followed by weakness of legs. This was soon followed by tingling numbness and pains in the legs, and lastly by œdema, not affecting feet. By advice of physicians, Mr. J. sailed from Para, October 27th, but instead of immediately improving, he grew worse. Soon noticed numbness and awkwardness of hands; the weakness and œdema of legs increased. In the last six days the long extensors and the long flexors of the right thumb have shown paresis, and in the last four days the same muscles on the left side have been affected. No dyspnœa, vertigo, or mental symptoms. No fever observed; urine high colored. The pains were never severe.

*Examination.*—Stout, well-built man; complexion pale, muddy, and tanned. No motor or sensory symptoms in eyes or face. Heart normal; no effusion in pleura. Upper extremities: all voluntary movements present except extension and flexion of phalanges of both thumbs; the two long extensors on both sides are completely paralyzed, and the long flexors partially so. The thumbs hang as do fingers in certain cases of lead paralysis. The left extensor indicis is weak. The abductor pollicis longus, and the small hand muscles are normal on both sides. Opposition well done. Supinators strong. No ataxia. Grasp: right and left, each 25 degrees on dynamometer. Sensibility is normal to æsthesiometer on finger-tips; but, by pricking, a limited area of analgesia (incomplete) is found over the left metacarpus, in the

ulnar territory. Temperature well perceived. Lower extremities: the walk is somewhat feeble, but not pathological in type; can stand upon his toes, and equilibrium with eyes open or closed is nearly perfect. No ataxia. Patellar reflex abnormal on both sides. Both legs present a soft œdema in their inferior half; the calves are large but flabby. No swelling distal of ankle-joints. All voluntary movements of legs, feet, and toes can be performed; though extension and flexion of toes are done feebly. The great toes are not as much affected as are the thumbs. Sensibility is slightly dull to touch (a numb feeling) as high as the umbilicus; but the only zone of actual analgesia is an outer (fibular) side of left leg in its lower third. Temperature well perceived. Urine is dense, deposits urates; without albumin; a few small hyaline casts. Summary of electrical examination: Arms, no faradic or galvanic nerve reaction in extensor primi et secundi inter-nodii pollicis on either side. These muscles do not respond to faradism, while galvanism yields a slow contraction, with marked tendency to tetanus:  $\text{CaCC} = \text{AnCC}$  or  $\text{CaCC} < \text{AnCC}$ . In flexor longus pollicis (both sides) there is no nerve reaction, and only a faint oscillatory C, with a strong faradic current applied directly to muscle. Galvanism gives a slow contraction:  $\text{CaCC} = \text{AnCC}$ , and the latter tending to tetanus. The long extensors of fingers on both sides yield reduced faradic contractility, and rather sluggish galvanic contraction, through  $\text{CaCC} > \text{AnCC}$ . Legs: Quantitative reduction to faradism and galvanism in calves:  $\text{CaCC} = \text{AnCC}$ . Extensor longus pollicis and extensor longus digit. have almost no faradic contractility, and to galvanism yield slow C, with  $\text{CaCC} = \text{AnCC}$ , the latter almost tetanic. Through external popliteal nerve better contractions (both by faradism and galvanism) are obtained in these muscles. Tibialis anticus and small muscles of feet, normal reactions. In other words, we have in this case a well-marked DeR, limited to the nervo-muscular apparatus of both thumbs; partial DeR in long extensors of fingers and in extensors (and flexors) of toes.

*Second Category: Analogous Indigenous Cases.*

*Case I.*—Mrs. X., aged forty years, seen in consultation with Dr. Carreau, of New York, in November, 1885. There is a clear history of excessive drinking and sexual indulgence for many years. Patient is still drinking brandy steadily. For more than two years has suffered from gastric catarrh and failing memory

Early in past summer felt burning sensations in feet, pains in feet and legs; occasional numbness in feet and hands. At end of July marked increase in gastric symptoms, diffused swelling of hands and feet with prickling numbness and a hyperalgesic state of same parts. Was unable to walk. No arthritis. Hands have also been weak. No bladder symptoms. Pains have ceased, but hands and feet are in state of painful formication; dreads to be touched. There is frequent vomiting of food and mucus. Partial dementia, with a few delusions. Urine examined several times; no sign of renal disease. *Examination*: Tongue tremulous and foul. No motor or sensory symptoms in eyes and face. The grasp is feeble, but long extensors and flexors all act. Opposition of thumb is impossible; marked atrophy of radial side of thenar eminences. Examination shows marked reduction in tactile sensibility in tips of all fingers. The hands are œdematous, with dorsum tense. No arthritis. Lower extremities examined seated: feet puffy and tense, but not red; no arthritis. All movements of feet and toes are possible, though feebly done. State of sensibility cannot be well studied, owing to patient's absurd fear of all instruments and manipulations. This also prevents any electrical tests. Patellar reflex abolished (?). Spine not tender. Diagnosis: alcoholic multiple neuritis. Treatment by reduction of brandy, injections of strychnia, galvanism, and massage begun. Carlsbad sprudelsalz for gastric catarrh. A note received from Dr. Carreau, a few days ago, states that faradism did not produce contraction at first, and galvanism was used alone until March, when both currents were employed. In March began to walk, and has steadily improved. Now well, except feebleness of memory, and some numbness.

It is interesting to consider that this and other alcoholic cases bear a certain etiological relation to tropical beri-beri. In both cases we have subjects living upon a relative or actual excess of carbonaceous food, alcohol, or rice, as predisposing, if not efficient causes of neuritis.

The two following cases were published in my essay on "Myelitis of the Anterior Horns" (1874 and 1877) as examples of that affection. Reading them in the light of recent advances in nerve pathology, they now appear to have been cases of multiple neuritis, with dropsical symptoms, making them strongly resemble beri-beri or kak-ke. I give them merely in epitome, referring to the original for details.

*Case II.* (Case XVIII. of essay).—Male, aged forty years, seen in January, 1874. In September severe "cold," with bronchitis, temporary suppression of urine, swelling of face and feet, lasting three to four weeks. Some stiffness and subjective coldness in feet. Relieved by strychnia. October 16th, feet heavy and cold, hands awkward and fingers numb. Increasing loss of power in legs. Physician stated positively that neither this nor the former attack was due to Bright's disease. In November marked anæsthesia and nearly complete paralysis of both legs; thigh muscles remained active; no rectal or vesical symptoms. Median distribution in both hands, anæsthetic; the hands and forearms rapidly wasted. No bedsore or cincture feeling. No cerebral symptoms. Improvement from end of December, when movement appeared in toes.

When examined by me he presented an atrophic paralysis of both hands, with partial anæsthesia (mostly tactile) of finger ends. Toes (except great toes), feet, and legs, can be pretty well moved in bed; legs much atrophied, especially in anterior tibial group of muscles. Feet are partly anæsthetic, but present (as do the legs) marked hyperalgesia. No faradic reaction can be had in both extensor pollicis, and only feeble contraction in thenar eminences. These muscles contract well, however, to galvanism (DeR). Complete recovery occurred by end of summer.

*Case III.* (Case XIX. of essay).—Male, aged forty-seven years, seen in March, 1884. Large, healthy man. Severe "cold" in October, 1883, with bronchitis, slowly followed by numbness and paresis of limbs. The numbness appeared at about the same time in the ends of the upper and lower extremities. November 10th, walking was possible only with the assistance of a stick or a person, but he could still dress himself. Swelling of the feet, with glossy skin, was marked; by November 14th, subjective coldness of feet. A few days later was unable to sit up in bed; much formication up to the knees, and half-way up the forearms; some anæsthesia (?) of the feet. No head or eye symptoms; bladder and rectum normal. In the middle of January, 1884, intense formication ushered in recovery; gradual improvement in power of legs and hands. Feet remained a little swollen. Some wasting of legs. In January noticed numbness in distribution of left superior maxillary nerve on face. Can now walk with help of cane and crutch.

*Examination.*—No facial symptoms. Movements of arms and

hands good, except that extension of the right fingers is incomplete, and is very feeble on left side, especially for the thumb and index-finger. Palmar muscles emaciated, but not atrophied. Sensibility good. Grasp : right,  $26^{\circ}$  ; left,  $16^{\circ}$ . Co-ordinates well. In lower extremities the only paralysis is below the knees. The left foot is but slightly movable ; the right, a little more. Toes are somewhat contracted in flexion. Sensibility to contact is good down to ankle, dull below that, very dull in toes. Toes are numb. No œdema of feet or legs. Urine is dense; but free from any signs of renal disease. Electrical examination shows very great loss of faradic contractility in palsied muscles, almost complete in left extensor indicis, and in extensors of toes and flexors of feet (anterior tibial groups) ; feeble reaction in calves. All these muscles, however, react well to galvanism (DeR.).

These three indigenous cases of multiple neuritis—one clearly of alcoholic origin, the others of unknown etiology—I submit with but little comment. They were characterized by a combination of paræsthesiæ and anæsthesia, by paralysis with atrophy and DeR., of irregular peripheral distribution, and by œdema of the extremities, without signs of renal disease. Bronchitis occurred a short time before the nervous symptoms appeared in the two non-alcoholic cases. Cardiac and respiratory symptoms, indicating neuritis of the vagi and the phrenic nerves, were the only ones wanting to produce a symptom-group almost exactly like that of severe beri-beri or kak-ke.

DR. H. C. WOOD made some remarks on

#### THE DIAGNOSIS OF NEURITIS

and showed two illustrative cases.

DR. CHARLES K. MILLS then read some remarks on

#### THE CONCURRENCE OF MULTIPLE NEURITIS WITH MYELITIS OR ENCEPHALITIS.

As one result of the interest which has been aroused in the subject of multiple neuritis, or polyneuritis, some cures have been effected which were before not considered possible ; but it seemed to the speaker that, as in the case of other advances in medicine, some have been led to go too far in their conclusions, and have overlooked facts which are worthy of close attention. That multiple neuritis frequently occurs, can no longer be doubted ; but that it occurs frequently as an absolutely isolated affection is, to

say the least, doubtful. His own experience was, that not infrequently either myelitis or encephalitis, or both, concur with this affection; that is, in not a few cases in which the diagnosis of multiple neuritis could be made by the strict rules applicable to the study of this affection—by a certain combination of motor and sensory symptoms, by their localization in distal parts of the limbs, and by tenderness of the nerve trunks, and of the muscles—positive evidences of the existence of the central disorders were also present.

If, as is generally believed, multiple neuritis is the result of a direct toxic influence on the nerve elements themselves, it was reasonable to suppose that this same influence would frequently be exercised upon the nerve elements of the brain and spinal cord.

Dr. Mills' conclusions were that we may have four classes of cases: 1. Cases of multiple neuritis, pure and simple; such cases, however, were rare. 2. Cases in which the multiple neuritis was associated with myelitis. 3. Cases in which multiple neuritis was associated with encephalitis. 4. Cases in which multiple neuritis was associated with both myelitis and encephalitis. Instead of the association being just as is indicated by this classification, the combination might be of neuritis or peri-neuritis, with meningo-myelitis or meningo-encephalitis; or, finally, any modification of these combinations might be found.

Dr. Mills referred to a case which he had seen in consultation recently, in which the patient presented all the typical marks of multiple neuritis, the condition of her limbs being such as to call to mind the picture in Gowers' recent book of a case of multiple alcoholic neuritis, with palsy of extensors of wrist and flexors of ankle. This patient had also very positive evidences of cerebral involvement—mental confusion, with hallucinations and delusions both of sight and hearing. She had a clear history of abuse, not only of alcohol, but also of opium, chloral, and other narcotics. He believed that in this case there was a concurrence of multiple neuritis with a similar pathological condition in the spinal cord and brain. He also referred to other cases, seen in consultation, and more particularly in the Philadelphia Hospital, where the majority of patients have been the victims of alcoholic excess—cases in which a confused picture of nerve, cord, and cerebral disease, probably inflammatory, was frequently presented. Cases of poisoning by lead, mercury, and particularly arsenic, were also discussed. Particular reference was made to the series of cases



of poisoning by arsenic which he reported to the College of Physicians of Philadelphia in 1883.

A most interesting case, as bearing upon the views here presented, was one which he saw with Dr. O'Hara, of Philadelphia, to whom he was indebted for notes of the past history of the case, and also for suggestions as to the condition present at different stages. This patient, a professional man of originally good physique, and of remarkable intellectual attainments, had, eight years ago, an attack of rheumatism, which left him with a crippled heart, the damage to this organ, however, not seeming to have been of its valvular, but rather of its nervous structures. The patient was in the habit of using tobacco and alcohol, but not to excess, and sometimes did not use these agents at all for long periods. Two years after the first attack of rheumatism he began to have trouble with his vision. Sight in both eyes slowly failed until he scarcely had more than perception of light in both eyes. Careful ophthalmoscopic examinations were made with negative results; the media were clear, and no signs of retinitis or neuritis were discovered. In eighteen months he entirely recovered his sight from travel, rest, and the use, in the latter months, of strychnia hypodermatically injected. About a year after recovery of his sight he had another attack, rheumatic or rheumatoid gouty in character. In this attack he had insomnia and evidences of general nervous irritability. At this time the left upper extremity suffered from an undoubted attack of neuritis, affecting the most of the brachial nerves, but more markedly those of the median distribution.

About six months ago, he had what appeared to be an apopleciform attack, his left side—face, arm, and leg—being temporarily paralyzed, but without unconsciousness. The attack passed off in a few hours.

About two months since he was engaged in work which called for both mental and physical effort, and considerable exposure. One day, when at a height of more than one hundred feet, he was suddenly stricken with incomplete paralysis of motion and sensation in the lower extremities, accompanied by great pain, but succeeded by his own efforts in getting again to the ground. This attack he attributed, probably correctly, to a stroke of air. On examination, he was found to have almost complete paralysis of the left leg from the knee downward, with blueness and coldness of the foot, and typical anæsthesia dolorosa as high as the ankle. He

complained of excruciating pains, which he described as "jamming" in the sole of the foot. The right leg showed a similar condition, but much less marked, the paralysis of motion and sensation extending only half-way to the knee, and not being nearly as complete as upon the other side. In two weeks, under the use of anodynes, local and general, with massage and weak currents of electricity, he recovered completely from these symptoms in his limbs. He then began again to use both head and limbs, without rhyme or reason, and was attacked with loss of power and great pain, and perverted sensation in the right leg below the knee, with swelling of the dorsum of the foot and of the ankles. He also began to have lightning pains, here and there, in both lower extremities, and radiating from the spine upward, these being sometimes accompanied by retraction of the head. Still later he developed symptoms of cerebral excitement—total insomnia, refusal to take medicine, quasi-delusions, great loquacity, etc. He was without sleep for nine days and nights. Various remedies were vainly tried, but the first genuine relief which he obtained was from hypodermic injections of morphia, repeated every twelve hours. This was followed by the administration of deodorized tincture of opium regularly every four hours, in doses of from ten to twenty minims, with twenty minims at bedtime. This treatment was continued, with digitalis, which he had been taking more or less for six or seven years, for about nine days. Mild alcoholic stimulus was also used, and nourishment was systematically administered. He has improved to such an extent that all the symptoms, neural, spinal, and cerebral, have greatly diminished, so that he is able to travel, but he cannot yet endure any great strain, either physical or mental.

DR. JAMES HENDRIE LLOYD read :

NOTES ON THE ELECTRO-DIAGNOSIS OF NEURITIS AND ANTERIOR POLIOMYELITIS.

The irritability of a nerve-trunk to an electric current (either by galvanism or faradism) depends upon the integrity of the axis-cylinder. This integrity may be impaired by pressure upon or by a fracture of the axis-cylinder. The pathology of degeneration in a nerve-fibre is, first, an increase of protoplasm about the nucleus in the sheath, then the splitting up of the white matter, and, finally, the breaking of the axis-cylinder. In neuritis it may be a question how soon this *fracture* takes place, but *pressure*

evidently occurs very early, and in severe acute neuritis (as in some forms of facial paralysis) this pressure is very great, is very quickly established, and, in the worse forms, no doubt leads to the breaking of the axis-cylinder. If electro-irritability, therefore, be retained in the nerve in neuritis, it must be either because the pressure is very slight or else because of an escape of some nerve-fibres amidst the destruction of others. The first event is what happens in mild subacute neuritis, but the latter alternative does not seem very probable when the disease is interstitial in the narrow calibre of a nerve-trunk, and especially when it has continued a long while. On the other hand, in anterior poliomyelitis of slow progress the large cells may be gradually and successively destroyed, followed by the degeneration of a comparatively few nerve-fibres—and in these cases the nerve will retain its electro-irritability (possibly diminished sometimes) although the muscles may have commenced to put on the modal and serial changes of degeneration. These facts have been long recognized, but do not appear of late to have been sufficiently relied upon as a possible factor in diagnosis.

De Watteville has drawn a distinction between myotrophic and neurotrophic centres in the cord, but the distinction, as well as his illustration, is artificial, and the phenomena probably depend upon the extent of degeneration and the number of nerve fibres involved. His own opinion inclines to the view that where nerve-irritability (especially faradic) is preserved, especially in cases otherwise chronic, with muscular atrophy and muscular reactions of degeneration, we have a condition rather of atrophy of the anterior horns than of the nerve-trunks, affording strong corroborative evidence in establishing the differential diagnosis. It is this condition of retained nerve-irritability, with degeneration reactions to galvanism in the muscle, which we have in such slow cord-lesions as amyotrophic lateral sclerosis and chronic anterior poliomyelitis. Here other symptoms aid the diagnosis, especially the large fibrillary contractions in the muscular atrophy and the spastic symptoms in lateral sclerosis. The only forms of neuritis that he knew of—in which faradic irritability of the nerve is preserved, with muscular reactions of degeneration—are essentially mild, non-chronic attacks (such as mild facial palsy), and in these the lesion is probably light compression, leaving the axis-cylinder unbroken.

It must be recalled that muscle-tissue alone will not respond to such strength of faradism as we use upon it in the clinic, but is

excited through the motor nerve-endings in its tissue, consequently when we have response in a muscle to faradism we infer that the nerve is still intact or partially so. Hence in severe facial palsy one of the first symptoms is loss of farado-contractility.

Illustrative cases were cited.

*Conclusions.*—The point sought to be emphasized is the retention of electro-excitability in the nerves, which in chronic cases does not appear consistent with the idea of a neuritis, but, rather, of a slow cord-lesion. If this electric excitability can be preserved in a chronic neuritis, we must suppose a very slow interstitial inflammatory process which compresses some fibres and allows others to escape. But to think that this happens in the narrow calibre of a nerve-trunk during a prolonged period of inflammation seems to require some effort of imagination and credulity. The electrical reactions in the above cases (especially the condition of *nerve-irritability*) support the other symptom-groups in their significance as to a cord- or a nerve-lesion.

DR. H. C. WOOD stated his belief that chronic multiple neuritis is one of the most frequent of the severe organic nerve diseases, and that in a large proportion of cases of serious nerve disease, the whole spinal axis and its accessories are affected. In general paralysis of the insane, four-fifths of the cases have some lateral or posterior spinal sclerosis, and a large proportion of these cases, no doubt, have multiple neuritis. He thinks that it is perfectly proven that locomotor ataxia may travel down the cord and produce peripheral neuritis, and that it is equally well proven that peripheral neuritis may travel upward, producing secondary lesions in the cord. With reference to electrical studies, it may be said that a large number of cases show that in the various forms of neuritis every possible change in the electrical reactions can be found. Post-mortem examinations are sufficiently numerous to show that peripheral neuritis is frequently not associated with other diseases, and that it frequently causes symptoms which simulate other affections. When we have peripheral neuritis, it is difficult to make out a spinal trouble back of it.

DR. OSLER thought that it was clear that we have the curious disease beri-beri in this country. It is probable that it exists in the Gulf States, as, a few months ago, he received some slides, which had been sent by a physician of Texas, and were said to contain the micro-organism of beri-beri. Any one reading the descriptions of this disease must have been struck with the

remarkable resemblance of some of the cases to acute ascending paralysis. His friend, Dr. Vineberg, now of New York, who formerly practised in the Sandwich Islands, gives a very accurate account of the suddenness of onset, the rapidity of the ascension of the paralysis, which, in some cases, ultimately involves the respiratory muscles, producing death in that way. He agreed with Dr. Wood that multiple neuritis must be an exceedingly common disease. The French journals of the current year contain a surprising amount of literature on the subject. He had been particularly interested in cases of neuritis occurring in connection with certain of the fevers. After typhoid fever it has been described a number of times, and also after phthisis. During the past year he has had one interesting case of paralysis of the extensors of the left foot in connection with phthisis, unquestionably a case of neuritis. He suggested that possibly some of these cases of paræsthesia of the hands, with feelings of numbness and tingling, which Putnam has described so accurately, may be of the nature of a peripheral neuritis. A case recently presented itself at his service at the Infirmary for Nervous Diseases, in which these symptoms were very marked, and in which there apparently were some trophic changes. The skin was glossy, and the hands were somewhat swollen, and, indeed they presented in a slight degree the appearances seen in myxœdema.

In connection with the diagnosis between acute ascending paralysis, anterior myelitis, and multiple neuritis, he would refer to an article by Francotte in the *Revue de Médecine*, to which attention was called in *The Medical News* of July 3d, 1886. He reports four cases, two of which were studied anatomically, and in which the cord was found to be absolutely normal. In these two cases, the clinical picture was that of Landry's ascending paralysis.

DR. SEGUIN said that he was much interested in the remarks of Dr. Mills as to the possible co-existence of a central with a peripheral lesion. He had no doubt that this co-existence is frequently a source of considerable difficulty in making up our minds whether a case is one of irregular poliomyelitis or of peripheral neuritis. It also seemed to him to be a matter of great practical importance as regards treatment that we should make up our mind whether we have a case of perineuritis or one of parenchymatous neuritis. The former disease, which is, he thinks, the more common, and perhaps present in some cases of so-called neuralgia, is

essentially an inflammatory change in the connective tissue, perhaps penetrating into the interior of the nerve bundles, but not affecting the nutrition of the axis-cylinder or myelin and not capable of producing any distinct paralysis or atrophy. The diagnosis may be made clear by relying on this distinctive point, that in interstitial neuritis there must be anæsthesia, even if limited in area; and a true paralysis and atrophy, even if restricted to a few muscles. In perineuritis the symptoms are more diffused, milder, more subjective, and do not present a specific appearance. He thought that Dr. Lloyd had struck the key-note in insisting on the importance of a careful study of the neural and muscular reactions. In true neuritis and interstitial neuritis, there must be degenerative reactions in some shape, distal from the point of disease, while in perineuritis and in some forms of myelitis the reactions are either irregular or simply a quantitative or nodal reduction.

The diagnosis is of great importance with respect to treatment. Perineuritis and meningitic conditions he treats with great energy with counter-irritation, with the actual cautery and blisters. Internally, he employs mostly tonic treatment, and does not resort to mercury or iodide of potassium unless there is a special reason for their use. Rest is also an important element in the treatment of perineuritic conditions. In essential neuritis, therapeutic measures can have but little effect, as we know that a natural repair takes place irrespective of artificial conditions. If we favor nutrition by our general remedies, that is the most that we can do, although galvanism may serve to keep up a better nutrition in muscles pending nerve regeneration.

In regard to the relation between neuritis and beri-beri, he did not wish to be understood as stating positively that we have beri-beri among us, but he thought that it would not be out of place to call attention to the analogy between the symptoms of the two affections. He thought it well to have the attention of physicians in our Gulf States called to the features of the disease. As the disease has recently appeared in northern Brazil, it may appear in our Southern States.

To be continued.

THE  
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Original Articles.

A CONTRIBUTION TO THE PATHOLOGY OF  
THE CEREBELLUM.<sup>1</sup>

By E. C. SEGUIN, M.D.

I VENTURE to publish this contribution, because it seems to me that a certain value attaches to cases which, fairly well observed during the patient's lifetime, are completed by autopsy. Especially is this true of cases of somewhat rare lesions, often of difficult diagnosis, as are tumors of the cerebellum. Even if the publication of such cases brings no immediate brilliant result, it may be of help, later on, to other workers in the same field.

The first case which I shall relate tolerably well fulfils these conditions: it was observed frequently, almost constantly, during eight years by several competent general practitioners and by myself; frequent notes of the symptoms were recorded; the patient was made the subject of clinical lectures at the College of Physicians and Surgeons in several successive winter sessions; and a correct diagnosis of the location of the lesion was made early in this period, being fortunately recorded in a letter written to the attending physician in 1878, seven years before death. Finally, a reasonably complete autopsy was made, which brought to light the lesion which had caused symptoms for so many years, besides recent lesions not at

<sup>1</sup> Read before the New York Neurological Society, Feb. 2d, 1887.

all connected with the almost pathognomonic symptoms upon which the diagnosis had been based.

Three other cases of tumor of the cerebellum are also briefly related and their specimens presented. They are not as instructive as is the first case, but they also seem to illustrate, in a somewhat useful manner, I trust, certain points in diagnosis, prognosis, and treatment. The four cases together substantiate in many ways the diagnostic laws first formulated by Nothnagel.<sup>1</sup>

CASE I.—P. M., a single man, 37 years of age, a retired officer U. S. Navy, was first brought to me by Dr. D. C. English, of New Brunswick, N. J., in the autumn of 1877. From numerous notes taken from that time until May, 1883 (when the last entry was made in my case-book by Dr. Amidon), I condense the following history of the case:

Illness began in 1867 (eighteen years before death) by much headache and an attack of partial loss of consciousness or powerlessness. Had several such attacks, never falling in them. In 1868, consulted Dr. Agnew<sup>2</sup> because of failing sight. Had no diplopia. A seton was put in the back of his neck, notwithstanding which headache continued as the chief symptom of the disease. He had no symptoms in the legs. Later, in 1869, the headache diminished.

In 1869-70, began to notice a gradually increasing loss of power in the legs; no pains in them.

Was in Europe from 1871 to 1874; legs and head troublesome; sight impaired, but not growing worse.

Returned home and was retired in 1874; has since been in *statu quo*. The head is perhaps less painful than in 1870.

In the last few months pain in whole of head, more in front; mostly in front of head and in orbits (none in face). This pain is not nocturnal, nor is it influenced by weather changes. No dizziness. Never diplopia or hemianopsia. No neural pains in limbs; hands skillful. Denies changes in speech and loss of memory. No difficulty in micturition. Most positively denies chancre and syphilitic symptoms.

Once in a while he has had a bilateral feeling of numbness of hands and feet, never permanent. This was not a numbness from position of limbs. Less headache. Has become pale and thin.

*Examination.*—The head is held stiffly and vibrates a little. The eyeballs show great horizontal nystagmus, especially when they are directed to his left. Pupils equal, normal, and of medium size. Ophthalmoscopic examination shows marked atrophy of both optic nerves (the left more), choroidal atrophy and abnormal

<sup>1</sup> "Topische Diagnostik der Gehirnkrankheiten," p. 78. Berlin, 1879.

<sup>2</sup> No record of this examination can be found.



pigmentation. O. D. V. =  $\frac{1}{100}$ , O. S. V. =  $\frac{1}{200}$ . There is no facial or lingual paresis or tremor. Speech is not perfect, but rather slow and hesitating; not exactly syllabic. The hands are steady and strong: R.  $50^{\circ}$ ,  $49^{\circ}$ ,  $45^{\circ}$ ; L.  $37^{\circ}$ ,  $38^{\circ}$ ,  $36^{\circ}$ , in three successive trials on dynamometer.

There is no ataxia in the gait, but the walk is peculiar: stoops much, staggers with feet apart and very active. With eyes closed the steps are smaller and more cautious, but the gait is not aggravated. Stands almost perfectly with eyes closed and feet slightly separated; but cannot stand with feet held together. The legs, in walking, are not jerked outward and forward, nor are feet brought down as in ataxia. Strength (resistance power) at knees normal. When walking with bare feet it is seen that the toes are abnormally active, clawing the carpet, as it were, to get support.

The sensibility of face, fingers, and legs (one tested) is normal.

There is slight ankle clonus (patellar reflex not noted).

I then made a diagnosis of disease of the cerebellum, excluding posterior spinal sclerosis.

After seeing the patient a third or fourth time, in May, 1878, I wrote as follows to Dr. English:

"Dr. D. C. English, New Brunswick.

"MY DEAR DOCTOR:—I have re-examined Capt. M. with great care, and can make out no material changes for better or worse. He seems not to be in a good general condition. I am now more certain in my diagnosis. I feel quite sure that he has disease of the cerebellum. According to very recent studies of cerebellar lesions by Prof. Nothnagel, of Jena, the disease in Mr. M.'s case is very probably central or bilateral and involves the vermis superior. I see no new therapeutic indication. It would be well to build him up for a while before resuming the former powerful medicines."

"Very sincerely yours,

"E. C. SEGUIN."

In the notes of May 14th, 1878, it is stated that lately there has been occasional diplopia for distance: that the patellar reflex is normal. The heart is healthy. The walk is again minutely described as *supra*.

Oct. 16th, 1878. Claims great improvement over last year's condition. No headache worth mentioning. Speech is perhaps better. Eyes unchanged. Walk is as before characteristic of cerebellar disease; with feet wide apart, arms used to keep equilibrium, toes clutching the floor. Co-ordination of hands perfect. Stands almost perfectly well with eyes closed. Speech thick and slow.

April 11th, 1879. Certainly no worse. Tongue deviates to right. V. as before. Grasp: R.  $50^{\circ}$ , L.  $40^{\circ}$ . Has a complex nystagmus. In central fixation, finger held directly in front of patient, it is downward  $\downarrow$ ; in outward fixation it is in either direction according to direction of the strain  $\rightarrow$  or  $\leftarrow$ . Has taken about one hun-

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<sup>1</sup> Courses of iodide of potassium and of nitrate of silver had been given.

dred grains of nitrate of silver since Feb'y 1st without discoloration of skin or mucous membranes.

October. No material change. Speech the same. Occasionally has had a feeling of impending loss of consciousness (*petit-mal*?). The patellar reflex is supra-normal on both sides. Slight diffused headache.

May 21st, 1880. No change except that nystagmus is different: constantly obliquely to left and downward ↙.

Oct. 27th, 1882. Last July noticed a peculiar sensation in epigastrium, not nausea. One day that month fell forward unconscious; there was no spasm and he did not bite his tongue. Since has had a "light" feeling in his head several times. This is first distinct attack since 1868. The walk is less good, but has same characters, groping, feet separated, hands reaching out, body bent forward. No true ataxia in hands or in feet (tested lying). Stands as well with eyes closed as with eyes open. Patellar reflex greatly exaggerated on both sides. Pupils active, of medium size; both optic nerves much atrophied, with small vessels. Pulse of normal rate, but over-tense. Urine recently re-examined and found normal. Manner slow, looks haggard. Sleeps well. At this time Dr. English thought that there had been no material advance in the disease.

In May, 1883, had nausea one day, ending in projectile vomiting lasting all evening. The next morning he was found semi-comatose, and became conscious the next day.

I did not see Mr. M. in the last two years of his life. He was attended a part of this time by Dr. D. C. English at New Brunswick, and by Dr. W. Elmer at Trenton during the last year.

Dr. Elmer has very kindly given me the following statement as to the course of events in 1885:

"On the 23d of February, after complaining of headache a few days previously, he was taken suddenly in the library with an epileptic convulsion lasting, if I remember rightly, about half a day before consciousness was regained. I do not think there was absolute paralysis of the left side at this time, but partial immobility; his power to move or lift his leg was quite limited, and he complained of pain in the leg and foot, and also in the hand, desiring friction of both members. The limb was not stiff, and yet motion was rather painful. The attack affected also speech and deglutition; the former being very indistinct and the latter performed with effort. I cannot say that the tongue was actually shrivelled, though I am sure there was no enlargement of that organ. Without any test by the *æsthesiometer*, there was hyper-*æsthesia* of the affected parts."

There was afterwards no sudden change in the patient's condition. New symptoms gradually appeared, such as increasing dulness, stupor, drawling, greatly impaired articulation, difficult deglutition, and polyuria; in brief, many bulbar symptoms. Con-

<sup>1</sup> Letter dated Trenton, Jan'y 20th, 1887.

vulsions occurred on the 20th of April, and death took place by coma on the 22d.

AUTOPSY.—On April 24th, 1885, forty hours after death, I made the autopsy in the presence of and with the assistance of Drs. English and Elmer. The following notes were taken on the spot by one of the gentlemen present:

The skull and dura mater are normal. The convexity of the brain is pale and rather flabby; here and there are opacities along the superficial vessels, and a number of these show streaks and patches of yellowish new-formation. In some, an old thrombus is demonstrable. Base of brain: olfactory bulbs are normal; the optic nerves small and of a dull yellowish color; the third nerves are also yellowish-white and dull-looking, but not small; the fourth, sixth, seventh, and eighth nerves appear normal. The same is true of the filaments of the hypoglossal nerves and of the lateral bulbar nerves. The carotids are stiff, gaping, and are the seat of marked arteritis of the patchy or nodose form (not calcareous). The branches of the carotids, the posterior communicating, and the posterior cerebral arteries show the same lesion. The basilar is more diseased, the arteritis occupying its whole length, making it a stiff, whitish tube with fair calibre. The vertebrals are less diseased, about as much as the carotids. No thrombi or emboli in any of the principal vessels.

Transsections of the cerebrum show dilated lateral ventricles. The nucleus caudatus is normal on both sides, but the section at the level of the chiasm shows the outer segments of both nuclei lenticulares in a state of softening; yellowish gray. The external capsule and claustrum are, however, normal. The caudal part of both thalami shows yellow softening, the left more than the right. The internal capsule looks white and normal.

Spinal cord: Its dorsal veins are extremely injected and varicose; the cord itself seems small for the size of the subject. A section through the cervical enlargement shows only a doubtful grayish appearance in the left postero-lateral column.

Cerebellum: On inspection, the caudo-dorsal aspect of the cerebellum reveals a large cyst, apparently two inches (50 mm.) in diameter, occupying the site of the vermis superior (middle lobe) as far as its frontal third, extending caudo-ventrad as far as the fourth ventricle. It appears that the vermis superior is destroyed, except its frontal third. The floor of the fourth ventricle is apparently not diseased. The cyst extends at least one inch (25 mm.) into the right lateral lobe of the cerebellum. The caudo-dorsal boundary of the cyst is simply a transparent membrane (arachnoid?). Its basal and lateral walls are thicker and somewhat gelatinous, but it is apparently a smooth membrane (no sarcomatous basis). The cyst is strictly monolocular and contains only a clear fluid without floating bodies.

The medulla oblongata and pons are both softer than normal, and the projection of the anterior pyramids and olives is somewhat

effaced. No sections were made, and the cerebellum attached to the pons and medulla was placed in a solution of bichromate of potassium for hardening. Other specimens were also preserved for future examination.

The heart showed marked hypertrophy without valvular disease. The aorta bore non-calcareous patches similar to those in the encephalic vessels.

The following is a fair summary of the case :

Beginning eighteen years before death with headache and one or more epileptiform or apoplectiform attacks. Impaired vision, with atrophy of optic nerves ; nystagmus ; continued headache, mostly frontal ; *typical cerebellar titubation* ; slight dysarthria ; no distinct paralysis, ataxia or anæsthesia ; patellar reflex exaggerated ; foot-clonus ; partial left hemiplegia without contracture. Death after cerebro-bulbar symptoms (stupor, greater dysarthria, dysphagia, salivation, and polyuria).

Lesions : Cyst of the cerebellum, destroying the middle lobe (or vermis superior), except its frontal third ; penetrating deeply into the right lateral lobe, but not destroying the nucleus dentatus ; exerting some pressure upon the floor of the fourth ventricle. Also, extensive arteritis obliterans, causing multiple foci of softening in the cerebrum. There was found, later, on making sections, a hæmorrhagic focus in the right ventral half of the pons (in the midst of the pyramidal fasciculi), causing descending secondary degeneration of the usual type.

ANALYSIS OF SYMPTOMS.—This long clinical history may, I think, be advantageously divided, for the purpose of analytic study in connection with the lesions found, into five periods :

1. The period of onset or of initial lesion. The account of this is very meagre, and we have no professional memoranda to guide us. Two or three attacks of an epileptiform or apoplectiform nature, preceded and followed by headache, marked the occurrence of the primary lesion or lesions. Quite certainly there was no marked paralysis or inco-ordination then, for the patient continued to perform his duties as paymaster, U. S. N. Without being at all positive in my assertion, I would suggest that these attacks represented hæmorrhages in the cerebellum, probably in the right hemisphere dorsad of the nucleus dentatus, and not far from the vermis superior. These hæmorrhagic foci, instead of cicatrizing, became fused in one active cystic formation, which gradually enlarged and

encroached more and more upon the medial part of the right lobe, ultimately involving the vermis, and exerting some pressure upon the floor of the fourth ventricle. The next year, 1868, the patient consulted Dr. Agnew for failing sight. In all probability, there was then the condition of neuro-retinitis passing into atrophy, so well known as a symptom of distending lesions in the brain. There were then no symptoms in the legs, so that if we rely upon cerebellar titubation as the special symptom of lesion of the vermis, the cystic or pre-cystic formation had not then encroached upon the middle lobe of the cerebellum, although it was capable of impairing the nutrition of the lobi optici, to such an extent as to produce partial loss of vision.

2. The period of impaired equilibrium, which extended from 1869-70 to within a few months of death. This was ushered in by "weakness" of the legs, followed in a year or two by staggering. In 1874, this disability had become so great that the patient was retired from the service. During the five years, 1869 to 1874, there were no new symptoms, and the eyesight, though impaired, did not grow worse.

It is very probable that in the years 1869-71 the cyst attained its maximum size and permanent relative position, as shown in the specimens.

3. A third and very long period of *statu quo*: the lesion quiescent, and no fresh cerebral lesions formed; from 1874 to within two months of death, let us say about twelve years. During eight years of this period, from 1877, Mr. M. was under my immediate observation. I saw him repeatedly, and three times at least made him the object, with his intelligent co-operation, of clinical lectures in winter sessions at the College of Physicians and Surgeons. He then presented the following symptoms: More or less headache, occipital and frontal, partial atrophy of both optic nerves, nystagmus of variable type, slight slowness and thickness of speech, and typical cerebellar titubation. Repeatedly was it demonstrated that there was no ataxia, strictly speaking, no paralysis or

anæsthesia. The mental functions were normal, and the special senses, except sight, active. The absense of fulgurating pains and the presence of good knee-jerks, excluded posterior spinal sclerosis, of course.

4. The attack of convulsions followed by partial left hemiplegia, which, according to Dr. Elmer, was purely motor and unattended by secondary contracture. This corresponds, I believe, to the hemorrhage in the right ventral area of the pons Varolii, and to the secondary descending degeneration which can be traced from it even with the naked eye.

5. The terminal period, characterized by many bulbar and cerebral symptoms. These symptoms were all due, in my opinion, to various localized ischæmias produced by the generalized arteritis, and not to any increase in the original cerebellar lesion. The arteritis, affecting as it did most of the basal and cerebral arteries, produced the various foci of softening in the cerebrum noted in the autopsy, and also led to impaired nutrition of the medulla oblongata. It may be a question whether the bulbar symptoms, dysarthria, dysphagia, salivation and polyuria, were all of strictly bulbar origin, or whether some of them may not have been the result of softening of the outer segments of the lenticular ganglia. Against this view, that we had here symptoms of pseudo-bulbar paralysis, may be urged the fact that the external capsules, claustra, and cortical substance were normal (to the naked eye, at least). It seems to me that the symptoms above named were truly of bulbar origin, though not due to pressure from the cyst. Sections through the medulla do not show any distortion of the floor of the fourth verticle, such as would have been produced by severe pressure downward by the cyst. The stupor and final coma, lastly, represent the more extensive softening in the brain, and its general ischæmia due to the arterial degeneration.

PATHOLOGICAL ANATOMY.—As described *supra*, this cyst was monolocular, filled with transparent fluid, and bounded by a definite limiting membrane all around. At the caudal end of the cyst, the membrane and pia mater

were united. A piece of the membrane from the basal part of the cyst appears firm, homogeneous in texture, and about the thickness of thick drawing-paper (about  $\frac{1}{2}$  mm.). It looks, as in the piece passed around, not unlike dura mater which has been hardened in bichromate solution and alcohol.

Fig. A represents fairly well the appearance of the cyst *in situ*, as seen from behind. It replaced the greater part of the caudo-ventral vermis, destroying more especially the pyramid, uvula, and the right tonsilla. Frontad, portions of the ventral vermis remained, overlying the fourth ventricle; this was made up of the lingula, as far as I could determine.

Figs. B and C are both made frontad to the greatest development of the cyst, and show it bounded, dorsad and ventrad, by more or less altered, foliated cerebellar cortex. The left nucleus dentatus is absolutely intact, the right almost normal in size, the lesion just reaching it. The gross specimen and the thin transsection passed around show the level of the floor of the fourth ventricle undisturbed; there was not, probably, any great degree of pressure upon it.

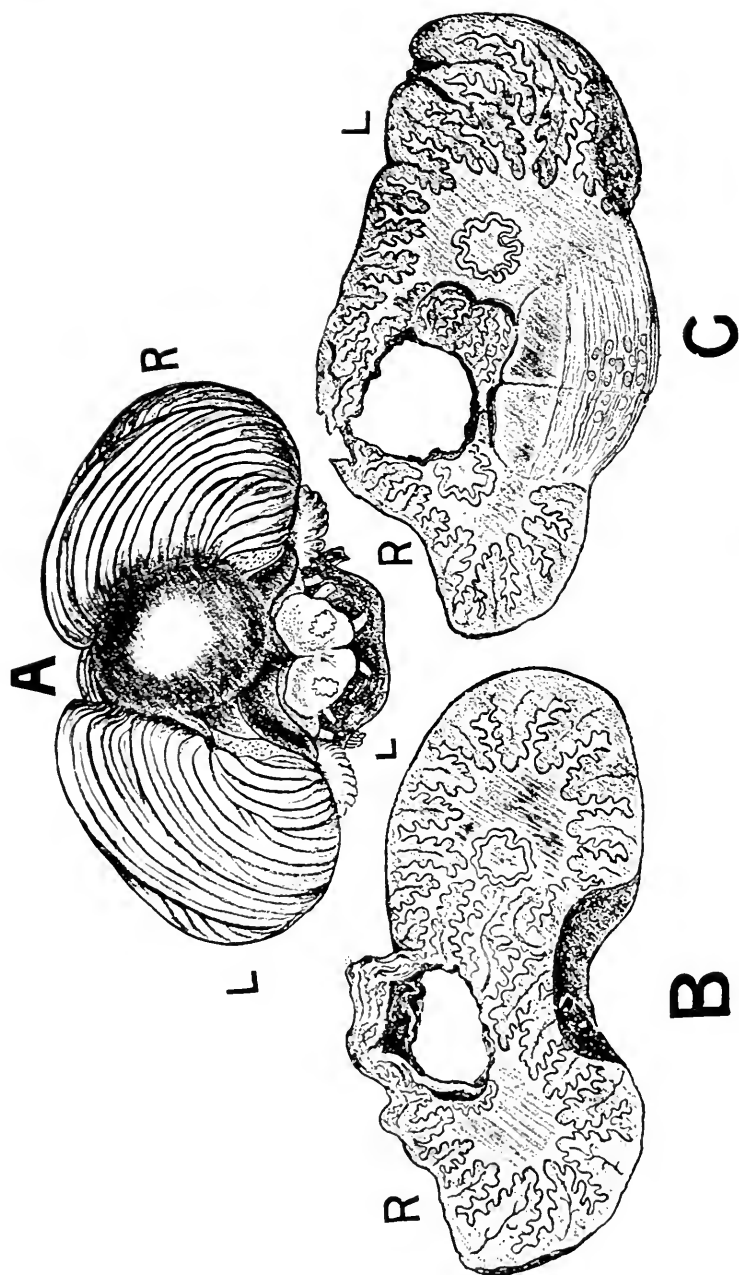
A series of transsections of variable thickness was made from a point just caudad of the frontal edge of the pons through to the post-optic lobes, and as far caudad as the level of the pyramidal decussation.

The spinal cord was found too brittle to permit of thin cutting, but pieces of it are shown exhibiting a distinct lesion.

The results of a study of this series of sections may be classified as follows:

#### A. Focal lesions.

1. A relatively recent focal lesion was found in the right ventral half of the pons frontad of the trigeminus level. This was a hemorrhage about 5 mm. in diameter, lying in the right pyramidal tract. Caudad of this focal lesion can be traced in all the sections, and in the fragments of the spinal cord, a typical descending degeneration of the pyramidal tract; in the pons and right pyramid, and in



Cyst of the Cerebellum. Case I.



the left crossed pyramidal fasciculus of the cord. The right anterior column also shows a tractus of degeneration near its edge. This lesion fully accounts for the attacks of convulsions and partial left hemiplegia which appeared two months before death. It is interesting to note that there was not a positive contracture of the paretic extremities to match this well-marked descending degeneration. I believe that occasionally degeneration of the pyramidal tract may exist without contracture, but in this case it might be said that death occurred too early to allow of the development of the contracture, as only eight weeks elapsed between the seizure and the fatal issue. The time necessary for the establishment of contracture in hemiplegia varies, from four to twelve weeks; still the fact that here we have fully developed degeneration of the pyramidal tract without contracture is interesting, and serves to throw additional obscurity upon the mechanism of hemiplegic contracture.

2. Changes in the medulla oblongata caused by disease of the arteries. There are histological changes in the nuclei of this region, but I have not had time to study them.

B. Secondary degenerations from the cerebellar lesion. This, it will be remembered, destroyed the caudo-ventral vermis and a part of the right hemisphere of the cerebellum, extending ventro-laterad as far as the nucleus dentatus.

1. The processus ad cerebrum (anterior peduncles of the cerebellum) appear normal and are equally developed as far frontad as their decussation point, just caudad of the nuclei tegmenti.

2. The processus ad pontem (middle peduncles of cerebellum) are normal; though their fibres pass through atrophied bundles of the pyramidal tract in the right half of the pons.

3. The processus ad medullam (posterior peduncles of cerebellum, more especially the corpus restiforme) appear somewhat reduced in volume in the bulbar sections on the right side.

4. The left olive is undoubtedly smaller, flatter, than its

fellow, and contains ganglion cells which seem smaller and ill-developed.

This atrophy of the olive is not at all extreme, not comparable with that shown me by the late Prof. von Gudden as resulting from removal of one-half of the cerebellum of a rabbit.

5. As far as can be determined in the imperfect sections obtained from the dorsal spinal cord, the ascending cerebellar fasciculi (direct cerebellar tracts) and the columns of Clarke are not atrophied or degenerated.

Consequently it may be concluded that in an adult man destruction of the caudo-ventral vermis (pyramid, uvula, and one tonsil), with some injury to the hemisphere of the cerebellum reaching to the nucleus dentatus, existing for about sixteen years, produces atrophic (degenerative) changes only in the corpus restiforme of the same side and the olive of the opposite side, both these organs being only slightly affected.

CASE II.—J. J., aged fourteen years. Seen first on July 29th, 1880. Had been a healthy boy. At three years had whooping-cough severely, with several convulsions. Parents deny convulsions or petit-mal since.

About January 1st, 1876, J. fell heavily on a stone walk, striking his head so hard as to make him unconscious; did not vomit. In April of that year he began to have curious vomiting spells in the early morning, followed by violent occipital headache. The patient describes the vomiting as not preceded by nausea, and the rejected matters contained no food. After having had these attacks for several days, one afternoon J. fell unconscious and had a general convulsion, repeated in the night. After this J. carried his head inclined to the left shoulder, his occipital headache continued, and he had a stiffish feeling in the neck. The vomiting did not return, and there was no delirium.

At the end of May he had gradually become paralyzed generally, but more on the left side. He had pain in his eyes with rapid failure of sight. Drs. Agnew and Knapp found white atrophy of the optic nerves. No recovery of sight since. (It is very probable that during April there had been choked disks, with fairly preserved vision.) Speech was never affected.

Spontaneous improvement occurred, and in July J. was able to sit up, and gained rapidly in all respects except sight. Some disability in use of hands and walking remained. He grew well, and was taught at a school for the blind. Has been very intelligent. No special symptoms occurred for nearly four years, viz.,

until May of this year (1880) when he began to have attacks of occipital pain and vomiting; occasionally had pain in left mastoid region, and numbness in left side of chin, and around left corner of mouth. A few days ago was found unconscious; probably having had a convulsion. Admits occasional dizzy or unconscious spells of momentary duration. Is still able to be up all day, dressed.

*Examination* (July 29th).—Eyes in left conjugate deviation; sightless; pupils wide; nerves bluish-white. Tongue straight; right hand  $20^{\circ}$ ; left  $25^{\circ}$ . Left leg stronger than right. Consequently has right hemiparesis; no tendon reflex at knees; walk is staggering, more off toward his left. There is no distinct ataxia, and the walk is not of the type called cerebellar; no anæsthesia. I gave him a mixture of bromide and iodide of potassium, of each salt about  $\text{℥}$ . at night; quinine, sherry-wine, and food.

Sept. 29th.—Patient improved wonderfully in first month of above treatment. Early in September had a sort of convulsion, and since more or less occipital pain; objective symptoms as above.

Nov. 14th.—Poorly of late. Occasional attacks of occipital pain and vomiting (without nausea); rather frequent attacks of pettimal, or perhaps more strictly speaking syncopal attacks, usually associated with headache. In last twenty-four hours has been semi-comatose, at times vomiting.

Nov. 16th.—To-day better, and is ordered ten drops of a saturated solution (equal parts) of iodide of potassium three times a day, to be increased each day by two drops at a dose. The small dose of bromide heretofore given (about  $\text{℥}$ .) stopped.

Dec. 6th.—The iodide has been gradually increased to forty drops three times a day, with the best results; no headache or vomiting or syncope since beginning iodide. No bromide. Rich food and sherry.

*Examination* shows a new symptom, viz., occasional twitching and distinct ataxia of the right upper extremity; none in the legs; perhaps a trace of ataxia in left hand. Absolutely no tendon reflex at knees. Right hemiparesis; no anæsthesia; face not paralyzed. Is up all day, and walks out-of-doors occasionally. Iodide to be gradually reduced.

Several times during the winter and spring of 1881, J. had a return of occipital pain and syncopal attacks; more recently of cervical pain also. These attacks were invariably cut short by blistering the nape of the neck or the mastoids, and by giving at once the full doses of KI, viz., from forty to fifty drops three times a day. Previous to Dec. 21st, the blisters had not been used, so that we may conclude that the more potent agent, in affording relief to the very distressing and threatening symptoms, was the iodide of potassium. The relief usually appeared in two or three days. Between the exacerbations the dose of iodide was from ten to twenty drops; and he had a variety of tonics.

The summer of 1881 was exceptionally favorable for J. He

was very well and happy. Though blind and slightly ataxic he enjoyed life, and was very cheerful. He had learned to do many delicate manipulations with his hands.

Oct. 12th.—J. was seized with convulsions, vomiting, and a gradually increasing pyrexia. Died comatose on 14th at midnight, with axillary temperature of  $103^{\circ}$  F.

Autopsy showed a tumor involving a large part of the inferior portion of the right hemisphere of the cerebellum, compressing the underlying portion of the mesocephale. The upper three-fourths of the same right hemisphere of the cerebellum was occupied by a cyst containing a clear fluid. The bottom of this cyst is the solid tumor referred to above. The cyst has disintegrated the upper and middle portions of the vermis superior.

The cerebral convexity showed abundant heavy patches of purulent sub-arachnoid meningitis, chiefly along vessels. The microscope showed in fresh serum preparations tubercle-like masses round about vessels, and at their bifurcation. This meningitis was the cause of death.

A microscopic examination of the cerebellar tumor showed the sub-cystic tumor to be mainly sarcomatous, cellular, and vascular, with foci of amyloid degeneration.

The family are all unusually healthy. Besides J. there are seven living children who are pictures of health. The father and mother are perfectly well, and always have been. The teeth of patient were normal, and he was a well-developed lad of rather hydrocephalic aspect. No suspicion of specific disease could be entertained in this case.

CASE III.—Paul K., aged eight years. Seen in consultation with Dr. Malcolm McLean, of Harlem, on Nov. 17th, 1879.

In the past eight or nine months has suffered from diffused headache, attacks of vomiting, double exophthalmus, and staggering gait. Has been seen by many physicians, most of whom attributed the symptoms to "malaria." Child grew steadily worse in spite of treatment on this theory, and in August was taken to the Catskill Mountains. While there seemed worse; headache severe; staggered and vomited; was very weak. In September came under Dr. McLean's care, with above symptoms; no paralysis or impairment of intelligence. Parents stated that there had been no epileptiform seizures and no fever. Small doses of iodide of potassium caused improvement. Treatment suspended in October.

In last two or three weeks again worse; severe headache, much of it occipital and frontal. Great enlargement of the head and separation of the sutures. Marked exophthalmus—staggering gait and pseudo-paraplegia. A few days ago, there occurred sudden recession of the exophthalmus, and simultaneously there appeared a soft, fluctuating tumor or swelling in the right occipital region.

There is no history of injury to the head, and no evidences of tuberculosis.

*Examination.*—Child pale and intelligent; speech normal; vision seems good by finger and color tests, but the ophthalmoscope shows double neuro-retinitis (choked disk) of moderate degree. No facial or lingual paralysis. Co-ordinates perfectly well. All the cranial sutures are wide open; anterior fontanelle closed; forehead not very prominent; no exophthalmus now. In the right occipital region, in the location of the lambdoid suture, is a soft, compressible subcutaneous tumor, walnut size, whose contents beat synchronously with the pulse. The appearance of this swelling caused a relief to all symptoms except debility. It might be supposed that this swelling contained fluid derived from the hydrocephalus, but from its location I felt considerable doubt as to this.

Patient walked feebly in a staggering way; no paralysis or ataxia. Not the typical cerebellar "titubation."

I made the diagnosis of internal cerebral hydrocephalus, probably from tumor of the cerebellum compressing the aqueduct of Sylvius. I advised against puncture and aspiration of the newly-formed sac, and recommended larger doses of potassium iodide.

Dr. McLean wrote me Dec. 30th of this year:

"We immediately increased the iodide of potassium from ordinary doses (5 to 10 grains) to 25 and 40 grains; so that he received amounts of the medicine varying from 90 to 150 grains per day. The medicine never disturbed his stomach, and his symptoms were certainly ameliorated by the larger doses, which were continued for four months without interruption. The pains in the head were undoubtedly controlled by the medicine."

The child died in the early spring of 1880. The autopsy was made by Dr. McLean, under difficulties; the examination being followed watchfully by relatives, so that it was hurried and no notes were taken at the time. As the tumor was enucleated for future microscopic examination, its relations can only be given approximately, as recalled by Dr. McLean, who has very kindly written me as follows: "It was situated in the left hemisphere of the cerebellum, bulging out toward the anterior and middle inferior lobes, and pressing upon the aquæductus Sylvii. It seemed to have made appreciable pressure on all portions of the left hemisphere, but was buried in the tissue of the lobes I have mentioned. There was considerable softening around the tumor. . . ."

The growth was a fibro-sarcoma. It was well that the externally protruding sac was not punctured, for it turned out to be the extruded lateral sinus.

CASE IV.—Doctor F., aged 28, seen at Morristown, N. J., with Drs. Stephen Pierson and Frankenhimer, Nov. 6th, 1878. An enthusiastic hard-working practitioner, who left Charity Hospital last year. Had suffered from some "pulmonary trouble" during the past two years (probably extensive pleuritic adhesions).

In September of present year (about two months ago), was attacked with repeated causeless nausea and vomiting. Soon after a severe pain, paroxysmal in character, showed itself in the range

of distribution of the right occipitalis major nerve. This was aggravated by movements. After a few weeks, the pain became occipital and non-neural (*i. e.*, not within the course of any nerve-trunk). The pain has been the chief symptom, though occasionally absent for a week or ten days. The present attack or recurrence of pain is the fourth; it is excruciating. Vomiting has continued more or less all the the time, and emaciation has progressed. No fever.

"He exhibited the ataxic gait for several months before he gave up practice at . . ., at times staggering in the street, and laying hold of fence-posts to keep him from staggering. He feared people would accuse him of drinking too much." [This is quoted from a letter Dr. S. Pierson was kind enough to write me Feb. 4th, 1887; it would thus seem that staggering was the *first* symptom in this case.]

Movement of the body and sitting up in bed aggravate pain. In the last two weeks there has been paresis of the right external rectus (sixth nerve), and diplopia. No hemianopsia. Has required a great deal of morphia and chloral to secure partial relief. There has been no fever, cough, or expectoration. No stiffness of neck or opisthotonus. Sight unimpaired. Has had no ordinary headache, and no dizziness. No bulbar symptoms. In last ten days both hands have been the seat of partial numbness (though not exactly as if parts were "asleep"). On 1st inst. (five days ago), Dr. Chas. S. Bull, of New York, found no lesion of the fundus; there was only a congenital excavation of the disks.

Syphilitic infection and injury to head positively denied.

*Examination.*—Patient a little sluggish from narcotics. The head is not tender. Paresis of right sixth nerve; third nerves normal. Disks distinctly choked with very tortuous vessels and minute hemorrhages in retina. Left cheek is perhaps paretic, but tongue points straight. The æsthesiometer shows marked anæsthesia: two points distinguished only at 40 to 50 mm. on the left side of forehead and left cheek. Finger tips are likewise affected, viz., on the right side points are differentiated at about 3 mm.; on the left at 5 or 6 mm. Simple contact and pinching are, however, well felt on face and fingers. Strength of grasp not tested. Patellar reflex absent. The patient was too feeble and wretched to be asked to rise and walk.

*Diagnosis.*—A tumor, probably tubercular, in the pons Varolii near the medulla oblongata. I was led into this error by the partial left hemianæsthesia.

The autopsy, performed by Dr. Pierson a few days later, revealed a solid (sarcomatous) tumor, occupying the greater part of the right cerebellar hemisphere to the median line; much pressure on adjacent parts.

I am indebted to Dr. Pierson for additional points of value in the history of this case.

DIAGNOSTIC CONCLUSIONS.—The symptoms presented by these four cases varied somewhat in their grouping, yet were singularly harmonious. Let us consider them in order of constancy:

1. *Lesion of the optic nerve*, either as choked disks or secondary atrophy, was present in all the cases. This is in striking contrast to my experience with strictly cerebral (hemisphere) tumors, in which the optic nerves were affected only three times in ten cases. There is reason to believe, furthermore, that choked disk is usually an early symptom of tumor of the cerebellum.

2. *Headache* was present in all cases. It was distinctly occipital and paroxysmal in cases II., III., and IV. Occipito-frontal and never severe in case I.

In case IV., the pain was at first for several weeks a neuralgia of the occipitalis major nerve on the same side as the tumor. This is rather difficult of explanation.

3. *Vomiting*.—This was a very early symptom in cases II., III., and IV. It was a causeless vomiting, occurring almost always in the early morning; and was usually accompanied by severe occipital, or occipito-cervical pain. Indigestion did not occur. I have recently seen two other cases (in children) presenting the unmistakable symptom-grouping of cerebellar tumor, in which inexplicable vomiting was the only symptom (eyes not then examined) for months. One little fellow had had his stomach washed out for supposed gastric catarrh for a period of at least two months, even after some distinctly cerebral symptoms had appeared.

In case I. vomiting was never (?) present.

4. *The walk* was affected in all cases.

In cases I. and IV., there was cerebellar titubation. This was typical in case I. In case II. there was simply staggering with tendency toward the left.

In case III. a diffused staggering, somewhat like that of alcoholic intoxication, was observed. It is to be observed that case I. is the only one in which the lesion destructively affected the vermis in its caudo-ventral parts. In the other cases the vermis was more or less compressed.

Consequently, case I. goes to support Nothnagel's law that cerebellar titubation is characteristic of a considerable destructive lesion of the vermis.

5. *Motor Eye-symptom*.—In case I. there were various types of nystagmus. In case II. there was conjugate deviation (without vision) to the left; that is, away from the lesion, contrary to what occurs in hemisphere lesions.

6. *Paralysis*.—Slight, but distinct right-sided paresis (not of face or tongue) was present in case II. The other cases presented only a diffused loss of power.

7. *Ataxia*, strictly speaking, was present only in Case II., affecting the right upper extremity. This, as well as the paresis, was on the same side as the cerebellar tumor.

8. *Anæsthesia* was found only in Case IV. It was demonstrable on the left side of the face and on left fingers (opposite the tumor).

9. *True vertigo*. Subjective or static vertigo was not present in any case. It has always seemed to me that this was not a strictly cerebellar symptom, but one indicative of irritation, direct or indirect, of the acoustic nerves. Probably static vertigo also occurs from disturbances in the hemispheric circulation, as in galvanization of the cervical sympathetic. It might be added that, in the two living cases of cerebellar tumor above referred to, vertigo is absent.

10. The *bulbar symptoms* shown at the close of life in Case I. were evidently due to malnutrition (ischæmia) of the medulla by reason of the arteritis obliterans which affected the vertebral and basilar arteries and their branches.

11. *Psychic symptoms* were wanting in all cases.

THERAPEUTIC CONCLUSIONS.—Three of the cases (I. II. III.) teach us that many of the symptoms of cerebellar tumor may be controlled, and the disease retarded materially by the free use of iodide of potassium.<sup>1</sup> The

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<sup>1</sup> Since the reading of this paper, a remarkable case of cerebellar tumor cured by iodide of potassium has been published by Dr. B. L. Milliken, of Cleveland, in the *Medical News*, February 12th, 1887. All the symptoms dis-



remedy should be given in full doses, irrespective of the patient's age, from 100 to 300 grains a day, largely diluted. Even the vomiting is sometimes controlled by this, as shown in my living case whose stomach had been previously washed out.

PROGNOSIS.—This is not necessarily fatal. Case I. died of an entirely different pathological process, viz., general arteritis obliterans and multiple softening of the encephalic mass. Case II. died of tubercular meningitis. In both these cases, the growth had been completely checked, and gave rise to few symptoms beyond impairment or loss of vision.

It is, therefore, reasonable to hope, especially in children, for an arrest of the cerebellar disease, with some residua, such as blindness and slight motor disability.

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appeared except atrophy of the optic nerves. Dr. Milliken makes no attempt to locate the tumor, but it seems to me quite clear that it was a cerebellar tumor, with another lesion of the right temporal bone (above ear).

# LOCOMOTOR ATAXIA. TWO CASES: ONE A CASE OF SO-CALLED SPINAL ARTHROPATHY; THE OTHER, ACUTE TABES DORSALIS.<sup>1</sup>

BY WILLIAM HENRY PORTER, M.D.,

PROFESSOR OF CLINICAL MEDICINE AND PATHOLOGY IN THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL; CURATOR OF THE PRESBYTERIAN HOSPITAL; ACTING PATHOLOGIST TO THE FOURTH DIVISION OF BELLEVUE HOSPITAL.

THE histories of the two cases were as follows:

CASE I.—Mrs. M. R., æt. 40, Germany, housewife, was first admitted to the Presbyterian Hospital September 11th, 1884. Family history negative. Patient commenced to menstruate at 16, and has been regular up to the last few years. At 17 years of age, she married a man who, she stated, “was after fast women.” Two years after her marriage, a “fever sore” developed on her lip, which did not heal readily, but did after local and internal medication. At about the same time she had a sore near the anus. This was followed by a sore throat, pain in the bones, and a falling out of the hair, but without an eruption. Patient never had a miscarriage nor a child. Four years after her marriage, patient had a cold and, with this blindness of the right eye, with ptosis.

Since 30, she has been a widow. Her memory appeared to be defective, and it was almost impossible to obtain a satisfactory history.

From the time her husband died until the early part of 1882, she was perfectly well; at that time she commenced to have recurring attacks of dizziness without loss of consciousness, and would fall backwards if unable to catch hold of something for support. The next symptom noticed

<sup>1</sup> Read before the New York Neurological Society, stated meeting, Jan. 4th, 1887.

was a feeling as if sand was under the eyelids; this lasted about two weeks, but rapidly disappeared under Prof. Roosa's treatment. About the same time her feet began to swell, the swelling gradually extending up to the knees. At this time she had a sharp pain in the precordial region which extended to the scapular region.

On account of great weakness, associated with nervousness, she was compelled to slide on the buttocks from step to step when coming down-stairs. There was a general feeling of pricking and a numbness which extended over the whole body. At times she would feel as if lost. Pains in the joints developed and were always increased prior to a storm.

When admitted to the hospital, the patient's general condition was poor; she was anæmic, fretful, childish, and unreasonable in her desires. Digestion good, bowels regular, but there was a constant desire to go to stool. Heart, lungs, temperature, and urine normal. Pupils persistently contracted to "pin-hole size;" they respond readily to distance, but not to light. There was a marked want of co-ordination in the muscular movements of the extremities, but without loss of power. There was a marked deformity of the left knee which consisted of a bulging on both sides of the joint, most noticeable upon the inner side. The prominence of the patella was absent. Manipulation showed that the superior extremity of the tibia could be dislocated in any direction. Accompanying the motions of the joint, there was heard a distinct crunching sound evidently produced by the rubbing together of the eroded bones which formed the articulation. An increase in the synovial fluid could not be detected. With all this swelling, deformity, and manipulation, there was a positive absence of pain. Before entering the hospital, the cavity of the joint was aspirated and four ounces of clear serous fluid withdrawn.

The right knee is somewhat deformed, but still maintains its contiguity.

Patient complained that she felt very nervous, and also of tingling throughout the body.

The pupils were dilated, and an ophthalmoscopic examination made. No changes were discovered except a slight hyperæmia of the disk.

The treatment consisted in the internal administration of iodide of potassium, strychnia, and ergot, with cups to the back and faradization to the extremities.

Both plaster of Paris and the straight posterior splint was applied to the affected joints, with a slight improvement in the condition of the arthritic condition.

The patient remained in the hospital until February, 1885, the symptoms fluctuating, sometimes being worse, at others decidedly better. The right knee-joint grew worse, and the crunching sound became quite apparent when it was moved.

When readmitted to the hospital in February, 1886, all her symptoms were decidedly worse. Her general symptoms improved for a time, but the joint trouble still persisted, but without pain, although she suffered considerably from the general ataxic pains.

On the 20th of August, diarrhœa occurred, accompanied by increased pain, especially in the back. This, however, was easily controlled. But from this time on she grew steadily worse. On the 26th a decided induration was detected on the left buttock, apparently about the size of a walnut, the surface around this point being quite red. The pain was very severe, and morphine had to be administered.

On the morning of August 27th, the temperature rose to 105° F. without any chill, vomiting, or headache. The ataxia became very marked in every movement. The phlegmon on the left buttock had a very red and angry appearance, and the induration was found to be extending, but the œdema was apparently confined to the cellular tissue.

At one point the skin was black, and looked like a gangrenous slough. A small opening was found near the centre of the phlegmon, and a probe introduced which led into several sinuses. Cocaine was injected, and the sinuses laid open and packed with gauze. Temperature

at midnight, 103.4° F. Thirty grains of antipyrin was administered at 2 P.M. Temperature was down to 102.2° F. Restlessness subsided, and patient slept well.

August 28th. Pulse 126; temperature still high. Aug. 29th. Temperature still higher. Pus flowing freely from the wound. August 30th. Midnight temperature 102.8° F. Antipyrin, 30 grains. 4 A.M. Temperature 100.4° F. Pus was freely excreted by the wound. Pulse remained weak. Patient had some vomiting and a cough with pain in the region of the sternum. Breathing good, but subcrepitant, and mucous râles were heard. Sept. 1st. Temperature still elevated, and the pulse continued weak, although stimulants were freely administered. Sept. 2d. Redness, œdema, and increased heat was detected over a spot about two inches square on the right buttock. The redness faded gradually at the periphery of the œdema, and appeared to be due to changes in the connective tissue without involving the skin.

There was marked cerebral disturbance and great thirst.

The temperature was partially controlled by antipyrin.

Sept. 3d. Iodoform and cold cream applied to induration on right buttock. Mental condition nearly that of delirium. No repair apparent in the wound of the left buttock, but the surrounding induration was diminished.

Sept. 4th. No improvement. Pulse very rapid and weak. Urine red in color and acid in reaction. Sp. gr. 1.030, albumin, pus, and casts.

Sept. 5th. Patient was very restless, and soon became delirious, steadily sank and died comatose at 2.30 A.M., Sept. 6th, 1886.

The *necropsy* was made thirty-one hours after death. Rigor mortis was marked. Body abundantly supplied with adipose tissue. No external mark indicating a syphilitic lesion was found. The knees were diffusely enlarged by fluid within the joint cavity.

*Thoracic Cavity.*—The *pericardial* sac contained one-half ounce of blood-tinged serum. The *heart* was enlarged and its cavities dilated. The left ventricular wall was thickened and its muscular fibres degenerated. Weight, twelve

ounces. The segments of the bicuspid and aortic valves were nearly free from any fatty infiltration. The *left lung* was slightly adherent to the chest wall at the apex. The *right* was quite firmly bound to the chest-wall throughout by old pleuritic adhesions. Both lungs were congested and œdematous, with a tendency towards a hypostatic pneumonia. Weight of left, 10 ounces; of the right, 16 ounces.

*Abdominal cavity.*—The *spleen* was about normal in its general appearance. Weight, eight ounces.

*Kidneys.*—Both glands were enlarged. The left weighed six and one-half ounces; the right, six ounces. Their capsules were slightly thickened and adherent to the underlying renal tissue; the surface of the glands being slightly rough after enucleation. Their cortex was thickened, pale, and granular, and the markings wavy. Microscopic examination showed the chief lesion to be located in the epithelial cells, which were quite granular; there was also a slight interstitial thickening. The lesion was the hypertrophic form of chronic diffuse nephritis.

The *liver* was slightly enlarged and fatty. Weight, sixty-eight ounces.

*Cerebro-spinal System.*—The meninges of the brain and spinal cord were slightly thickened, and the pia mater of the brain was unusually pale and contained numerous thickened patches. The spinal cord was unusually soft at the time the necropsy was made, apparently from post-mortem change. But even in this softened condition, the wedge-shaped connective tissue development in the posterior columns was quite marked. Owing to the softened condition of the cord, it was difficult to properly harden the cord. The sections, however, which were obtained by Professor C. L. Dana, showed beyond doubt that the lesion was one of advanced tabes dorsalis.

The knee joints were opened and found to contain a large quantity of thick grumous pus which had distended the synovial sacs to such an extent as to cause rupture in both joints. The pus of the joint cavities had found its way into the cellular tissue and intermuscular planes and accounted

for the diffuse purulent infiltration of both thighs and the phlegmons found at the buttocks, the septicæmia, and death.

There were also extensive articular changes. There were from fifteen to twenty intra-articular cartilages in each joint cavity, some of which were attached to the synovial cavity by a small pedicle of fibrillated connective tissue, while others were loose and free in the cavities. Microscopic examination showed that they were composed of a dense fibrillated connective-tissue stroma, thickly interspersed with cartilage-cells. The articular surfaces of the bones entering into the formation of the knee joints also presented extensive change, the cartilage of incrustation being almost entirely destroyed, the underlying osseous structures having undergone extensive rarefying and formative osteitis with the development of small irregular, eburnated articular surfaces to replace the lost cartilaginous coverings. Between the projecting eburnated articular prominences, there were found numerous excavations evidently due to a carious process or a rarefying osteitis.

CASE II.—Mr. H. H., æt. 53, Austrian, widower, laborer, United States six years, was admitted to the Presbyterian Hospital August 26th, 1886.

*Previous History.*—The patient says that he was perfectly well and had never been sick until six weeks prior to his admission to the hospital, at which time he had peculiar sensations in his lower extremities, sometimes pricking, sometimes sharp shooting pains lasting only a short time, then disappearing to recur again and again. Has the same trouble in his arms and hands. Throws his feet up at the toes when he walks; ankles quite stiff. Cannot stand steady with his eyes closed, complained of a sensation in his feet as if walking on sand; this feeling is continuous.

*Physical examination.*—Apex beat of the heart in the fifth intercostal three inches from the median line; apex beat very feeble. A systolic murmur was heard at the apex and it was transmitted to the left. There was a diastolic murmur over the aortic valve, and it was transmitted downwards. Liver and spleen not enlarged. Trip-ham-

mer pulse, visible pulsation of the brachial and ulnar arteries. Absence of patellar reflex. Marked ataxic gait, accentuation of the heel movement in walking. Urine acid, specific gravity 1.025, no albumin and no casts.

Aug. 30th.—Patient had considerable pain, especially in the legs. Weight, 133 lbs.

No special change up to Sept. 10th. During the night he was apparently delirious, as he made several unsuccessful attempts to get up and dress. Sept. 11th his locomotion had become progressively worse, so that he can scarcely get about the ward without assistance.

During the day he became semi-stupid and lay in bed most of the time.

Sept. 12th.—During the past night his temperature, which had been previously normal, suddenly rose to 105.5° F., he began to cough and appeared to have considerable mucus in his bronchial tubes, which he could not raise. The pulse rose to 135 per minute and the respirations to 40, accompanied by a decided condition of stupor. Some crepitant râles were heard at the base of the right lung, and loud mucous râles all over both lungs. The vesicular murmur was roughened and high-pitched.

Stimulants were freely administered without effect.

Late in the evening he became semi-comatose, and muscular twitchings, especially of the arms, developed. They soon became violent enough to amount to general convulsions. Pupils equal; no signs of a cerebral hemorrhage or emboli.

Toward the morning of the 13th he became unable to swallow. He passed urine freely. The temperature remained high and he died comatose at 5 P.M. Sept. 18th, 1886.

*Necropsy*, forty-eight hours after death. Body fairly well supplied with adipose tissue. No decomposition changes, the body having been kept on ice. Muscles dark in color. Both legs between the knees and ankles were the seat of numerous white cicatrices the margins of which were raised and deeply pigmented as if of syphilitic origin.

*Thoracic cavity*.—The *pericardial sac* contained two



drachms of clear straw-colored serous fluid. There was one large milk patch upon the anterior surface of the right ventricle.

The *heart* was slightly enlarged, but its muscular substance was pale, soft, and flabby, and its cavities dilated. Springing from the acute margin there was a marked adipose fringe, and there was considerable adipose infiltration between the muscular planes. The mitral valve was but little changed by fatty infiltration. The segments of the aortic valve were extensively thickened and retracted by fatty infiltration. This condition, together with the dilatation of the left ventricular cavity, perfectly explains the aortic and mitral regurgitant murmurs heard during life. The muscular fibres showed fatty degeneration.

The *left lung* was quite firmly adherent to the chest-wall at the apex, and to the diaphragm at its base. The inferior lobe was the seat of numerous zones of lobular pneumonia in the stage of red hepatization, the remaining portion of the lung being congested and œdematous. Weight of left lung, twenty-eight ounces.

The *right lung* was quite adherent throughout by old and fibrous pleuritic adhesions. The inferior lobe showed still more marked evidence of lobular pneumonia, a large number of lobules being implicated, and some having reached the stage of gray hepatization. The remaining portions were congested and œdematous. Weight, thirty-three ounces.

*Abdominal cavity.*—The *spleen* was small, soft, and had a large fibrous patch upon its convex surface. Perisplenitis syphilitica. Weight of organ, five ounces.

*Kidneys.*—The left gland weighed six (6) ounces and the right five (5) ounces. Both kidneys showed distinctly the foetal markings or lobulations. Their capsules were normal in thickness, and non-adherent to the underlying renal substance, the surface of which was perfectly smooth after enucleation. Their cortex was thickened and pale, but the markings were straight. The kidneys were practically normal. The *bladder* was over distended with urine.

The *liver* appeared to be normal. Weight, sixty ounces.

*Cerebro-spinal canal.*—The meninges of the brain and cord were thickened, especially the former. The dura mater was very firmly adherent to the pia mater and convexity of the brain along the margins of the longitudinal fissure. The pia mater was decidedly thickened and opaque throughout, numerous thickened patches also being found. No lesion of the cerebral substance was detected upon macroscopic examination.

When first removed, the spinal cord was firm and did not show any marked lesion, but after remaining a few days in the hardening fluid, it was plainly evident that there was a marked development of new fibrillated connective tissue in the posterior columns. Microscopic examination showed an advanced *tabes dorsalis* in the shape of atrophied nerve-fibres, and a decided increase in the fibrillated connective tissue of the posterior columns; this opinion being substantiated by the examination of the president, Professor Dana, who also examined the cord.

The two cases are interesting, as they illustrate a case of long duration with a complicating joint lesion, and one of unusually short duration.

The extensive suppurative arthritis without any pain directly referable to the joints involved is interesting.

The exact relation between the joint affection and the lesion of the cord is important. From the fact that joint affections are not, as a rule, associated with *tabes dorsalis*, it appears reasonable to look for the cause outside the lesion in the spinal cord, and to consider the joint affection more in the light of a coincident affection. As syphilis is a frequent cause for locomotor ataxia, and also for joint lesions, it seems reasonable to consider the syphilis as the etiological factor for both. The history, although not absolutely positive, points strongly in favor of a syphilitic infection a number of years back. The peculiar joint conditions are not those of an ordinary arthritis, but more strongly resemble the syphilitic lesions of the bones, in which there is a great tendency to have

both the formative and destructive process side by side. The changes are similar to the syphilitic osteoplastic periostitis with caries superficialis seen upon the surface of the long bones. It would appear, therefore, that the only connection between the two lesions was, that they both had the same cause.

It also shows that death was not caused by the cord lesion, but by the bursting of the capsules of the joints and the development of a diffuse suppurative cellulitis and septicæmia.

Exception may be taken to the short duration of the second case, but the history furnished by Dr. Alfred Edwards Hooker, house physician to the hospital, is undoubtedly correct.

It must be admitted by every one familiar with the pathological histology of posterior spinal sclerosis that a considerable development of new tissue and atrophy of the nerve fibres can be developed before symptoms are manifested.

In this particular case, the man always having been well and free from pains and aches, would be the one most likely to know had he suffered from them before his active symptoms developed. By a process of forced questioning, a series of symptoms might be developed which could be turned to account in making the history of longer duration. But in this instance even that resulted in a failure.

Although the patient's ataxia was rapidly progressing, his death was not directly attributable to it, but rather to the degenerated condition of his heart, and an acute broncho-pneumonia.

There appears to be no good reason why a patient could not develop some acute and complicating disease when the symptoms of locomotor ataxia are commencing and thus cause death early in tabes. Because a patient has symptoms of posterior spinal sclerosis for a few weeks, and then develops a pneumonia and dies is a poor reason for insisting that he must have had symptoms for a year or two without knowing it.

In fact, it is pretty generally admitted that locomotor ataxia in itself rarely, if ever, causes death ; but death, when it does occur, is always the result of some secondary and complicating condition. Why might not the complication be developed in the first week of the ataxia, and in this way render the disease of short duration?

## NOMENCLATURE IN PSYCHIATRY. MONOMANIA OR OLIGOMANIA, WHICH? PARANOIA, WHAT?

BY R. L. PARSONS, M.D.,

GREENMONT-ON-THE-HUDSON.

THE term Monomania was first adopted by Esquirol as a designation for certain phases of insanity which he had differentiated from the forms previously recognized. His differentiation was in the main good, but the term chosen was unfortunate for the reason that its very definite signification does not correspond with the idea he intended to convey. Hence the term was misleading from the first, so that he was obliged to undertake explanations which would have been unnecessary if the term had meant what he intended to express, or even if its meaning had been ill-defined or obscure.

The term was so misleading, in fact, as to lead Esquirol himself into errors and inconsistencies in the course of his descriptions of the disease; for while, in reply to the objections that there are no monomaniacs, that there are no insane persons whose reason is sound except on a single subject, that these patients always manifest some disorder of sentiment and will, he replies that *if it were not thus monomaniacs would not be insane*, he, on the preceding page, after stating that monomania is characterized by a lesion of the intelligence, affections, or will, goes on to say that at one time the intellectual disorder is confined to a single object, or to a limited number of objects, while at another monomaniacs are not deprived of the use of their reason, but their affections and dispositions are perverted, and in a third class of cases a lesion of the will exists, thus limiting

the aberration to a single faculty of the mind, or even to a single delusion of the understanding. And yet his very frequent use of the term *partial* in connection with this type of insanity, and a study of the cases he adduces in illustration, show conclusively that for the most part he thoroughly recognized the fact that the aberration in question did really extend to various faculties and at least potentially to more than one object.

If the use of the term *monomania* was unfortunate and misleading in the first instance, its use has since then become still more objectionable for the reason that the term as now used does not even mean what Esquirol intended. He says that the monomaniac is gay, petulant, rash, audacious, talkative, blustering, pertinacious, and easily irritated; nothing would appear to oppose the free exercise of all his functions. He contrasts him with the *lypemaniac* as his opposite in the state of his feelings. But the monomaniac, as now understood by those who use the term, is oftentimes depressed in mind and hindered in his mental operations. He writes that the course of monomania is more acute, its duration is shorter, and its termination is more favorable, unless there are complications, than in the case of *lypemia*. This certainly is not true of the typical monomaniac as now classified and described.

A monomaniac is generally understood to be a person who is insane in regard to a single subject only, while in all other respects his mental faculties are entirely sound. The obvious meaning of the term tends to establish this belief. As a natural inference, it is thought that monomania is not a very serious form of insanity, that the monomaniac is only a little insane. Lawyers and practitioners of medicine generally hold this view. The fact is, however, that the mental aberration designated by the term *monomania* is very grave in character, both in its medical and in its medico-legal aspect. This form of insanity is essentially chronic in its nature, and so the prospects of recovery are not as good as in cases of acute mania, or even of melancholia. Because of the apparent soundness of their reason in most regards, these patients

are liable to be considered as having a greater power of control over their feelings and acts than is really the case, and so their legal responsibility is liable to be over-estimated.

A careful study of cases of this form of insanity in which the aberration seems to be most restricted and of the simplest character will serve to show how profound, how fundamental the derangement actually is; and also in what respects certain typical cases differ from the description given by Esquirol.

Systematic writers on the subject of insanity express analogous views regarding the grave character of this form of mental aberration. Griesinger writes as follows, to wit: "Thus the excitement of the monomaniac does not pass so immediately towards the exterior; effort is accompanied by clear, conscious thoughts and opinions, loses thereby its instinctive character, and becomes actual morbid volition. With far greater, sometimes with perfect outward calm, there is a more profound internal loss of reason than in mania, because consequences soon result from the general excitation which set aside the essential conditions of healthy mental action." Maudsley writes as follows, to wit: "The course of monomania is not often toward recovery. The reasons are plain: in the first place, when it is secondary to mania or melancholia it signifies a chronic morbid nutrition which is a further stage of degeneration of the delicate organization of mind; in the second place, when it is primary, it is the morbid outgrowth of a fundamental quality of character, so that to get rid of it would be to undo the very character from its foundation."

The objections to the term monomania are such that many physicians engaged in the care of the insane do not use the term at all. An examination of the reports of fifty-one asylums for the insane, taken at random, shows that of the whole number of patients enumerated less than two per cent are classified as cases of monomania, while in twenty-four of these reports the term does not appear. In the reports for the Pennsylvania Hospital for the In

sane, thirteen per cent of the patients are classified as cases of monomania, while in the reports of the New York City Lunatic Asylum only fifteen-hundredths of one per cent are thus classified. If the reports of the Pennsylvania Hospital for the Insane had been left out of the account, the cases of monomania would have been reduced to less than seven-tenths of one per cent. Thus it would appear that even when the term is employed it is done without uniformity and without any reasonable approach to scientific accuracy, for such cases as were classified by Dr. Kirkbride as monomania undoubtedly exist in a similar ratio at other asylums.

Systematic writers of eminence on the subject of insanity also object to the use of the term. Tuke writes as follows, to wit: "We heartily wish 'monomania' had never been introduced into psychological nosologies, for if understood in a literal sense, its very existence is disputed, and if not, the various morbid mental conditions it is made to include by different writers leads to hopeless confusion. With one author it means only a fixed morbid idea; with another only partial exaltation; while a third restricts it to a single morbid impulse. As we proceed we shall consider its signification, but shall not frequently employ the term."

Dr. Sheppard, in his classification of the forms of insanity, writes of monomania (so-called), and approvingly quotes the following from Dr. Maudsley, to wit: "It is doubtful whether there is *ever only one point* on which the mind is unsound."

Dr. Maudsley writes as follows, to wit: "When the monomaniac (so-called) comes under the observation of one who is not only competent to observe, but has sufficient opportunities to do so, it will commonly be found that there is a bluntness or loss of his natural affection and social feelings, in consequence of his being so entirely centred in his morbid self; that his character and habits have undergone some change; and that he exhibits an excitability of mind with loss of self-control in circumstances which would not formerly have provoked it."



Dagonet writes as follows, to wit: "In fine, the term monomania might without inconvenience disappear from science where it becomes a cause of confusion and embarrassment in the study of pathological facts."

Sankey, as quoted by Spitzka, writes as follows, to wit: "The popular opinion about the existence of monomania, I need scarcely add, is a very erroneous one. The French writers use the term in a much more restricted sense; but to avoid confusion it is better to avoid the term altogether."

Morel, after quoting Esquirol's description of monomania as pertaining to a single idea or a single faculty of the mind, continues as follows, to wit: "We cannot too strongly invite the attentive reader to reflect upon these peremptory passages and consider whether Pinel and Esquirol, who wrote them, should not have arrived at the conclusion that, through an unfortunate confusion of ideas, they mistook a systematized delusion for an exclusive and local delusion."

"We affirm the close connection, the solidarity of the ultimate relation between the various acts of the intelligence, not only in the home of our observations and of our personal inductions, but also in the name of the history of philosophy."

"This being granted, the question is whether the condition of mental alienation can break this essential law of the unity of intellectual life; for it is clear that if logic and experience constrain us to decide this problem in the negative, we ought also to reject the theory of Esquirol. We could not have a complete idea of the motives which impel the insane to some of their acts, unless we were freed from error in regard to *monomania*."

But yet, however much systematic writers on the subject of insanity deprecate the use of the term monomania, they rarely succeed. There are manifestations of insanity which are neither melancholia nor dementia, but which differ so much from mania that another designation is required for them, and the objectionable term monomania is the one generally employed. In saying this, it is not

forgotten that there are some modern authors of eminence, as Hammond and Spitzka, who adopt the term without dissent. The latter author even undertakes an elaborate defence of the term, but this defence ought perhaps to rank rather as an apology. He writes as follows, to wit: "If words were to be eliminated from the vocabulary because they do not literally correspond with their acquired and accepted meaning, more than half of those in the medical dictionary, and about nine-tenths of those employed in the special branch of mental medicine, would have to be replaced by new ones."

The objection to the term, however, does not lie in the fact that its literal meaning and the signification attached to it by learned writers on the subject of insanity fail to correspond, but in the fact that its literal meaning is so well defined and so easily understood that it involves within itself an idea at variance with its real scientific meaning; and hence that its literal meaning is understood instead of the real one by most persons who see or hear the term. Even alienists, as we have seen, have a feeling that they are unconsciously influenced by its form to a misinterpretation of its meaning, so that many of them are disposed to avoid its use. The misleading of the term is liable to be of especial disadvantage in courts of justice, where the correct definitions of the learned counsel and of expert witnesses on one side may fail to enlighten the intelligent jury in opposition to the interpretations of counsel on the other, aided by the evident, implicit meaning of the term.

Now, it being granted that the term monomania is open to serious objections, the question arises whether it has become so identified with the subject of insanity that it must be retained as a sort of necessity; or, if this is not the case, what suitable term can be best used as a substitute.

It is evident that the term is not an absolute necessity from the fact that so many discard its use, although there are certainly strong reasons in favor of a suitable substitute. It is believed that such a substitute is available. In the annual reports of the New York City Lunatic

Asylum, the term monomania does not appear, although up to and including the year 1878 one thousand nine hundred and forty-five out of ten thousand one hundred and thirteen patients are classified as cases of mania partial. The designation mania partial, or partial mania, is a very good one in some respects. But it lacks certain requirements which a term suitable to designate important and well-marked characteristics of the disease should have. Such a term should consist of a single word, in order that it may serve as a basis for the classification of sub-varieties. In addition to this, the term should not have a well-recognized meaning of its own at variance with the idea it is intended to express; and it should not be already in use for some other purpose. The term oligomania is believed to fulfil all these requirements. Moreover, it is believed to be especially suited to replace the term monomania in the nomenclature of insanity, and hence it is proposed as a substitute for that term. The derivative meaning of oligomania is so obvious that no explanation is required in this regard. Its technical meaning, however, should not be too strictly inferred from its literal meaning, but should depend also, as in the case of other scientific terms, upon the definitions, distinctions, illustrations, and explanations attached to the term.

Within the past few years, the term paranoia has been used to a considerable extent as a substitute for the term monomania, especially by the younger writers on the subject of mental diseases. This fact is a still further illustration of the prevailing tendency to avoid the use of the term monomania. But the objections to this substitute are hardly less weighty than those which apply to monomania itself. If the meaning of monomania is too narrow for the purpose required, that of paranoia is too broad, and it may be added, too definite for the designation of something different from its evident meaning, which is simply distraction, craziness, insanity.

Griesinger writes that Heinroth has described a form of monomania in great part under the name of *ecstasis paranoica*, but does not adopt the term. Feuchtersleben

mentions the following as synonyms of folly, or insanity in the more restricted sense: "Insanity, according to Heinrich; dementia, bewilderment, according to Ideler; polymania, according to Fantonetti; paranoia, according to Weiss; and says that it proceeds from delirium as its highest degree." Feuchtersleben further says: "Reil seems to me to have given the best description of this form, as it occurs in nature, without pretending to state its essence. 'Fools,' says he, 'have no ruling idea: they change with their conceptions, and combine, in all sorts of ways, follies and eccentric tricks. Besides their general craziness there is a remarkable weakness of all the powers of mind, especially of the judgment.'" Paranoia as the synonym of folly here retains its original signification, and has nothing in common with the meaning to be conveyed by the term monomania.

Hughes makes use of the term paranoia as a synonym for primary monomania, Kiernan as synonymous with *monomanie systematisée*, monomania of Spitzka, primäre Verruecktheit of the Germans; Mills as synonymous with delusional monomania and also with moral insanity, thus giving a new and not very well defined meaning to the term, albeit one not in accordance with its derivative meaning and its classical use. The term paranoiac has also been employed, especially to designate what is popularly understood as a "crank." In fact, the general idea of a partial and ill-defined subdivision of oligomania would seem often to be in the minds of those who use the terms paranoia and paranoiac. Even in this case it is submitted that oligomania would be a better term, with such qualifying additions as might be required to express the exact idea intended, as primary oligomania, secondary oligomania, delusional oligomania, moral oligomania, mysophobic oligomania, systematized oligomania, oligomania of suspicion, or such other modification as might be needed.

In conclusion, then, with the assumption that monomania, as defined and explained by certain writers on psychiatry, designates phases of insanity of sufficient importance and well enough differentiated to require a place in the general

classification of mental diseases; and with the further assumption that the reasons adduced in this paper are sufficient to justify the substitution of the term *oligomania* for monomania, it only remains to apply the proper meaning of the latter term to the former, to wit: *A form of insanity which, although potentially affecting all the mental faculties and operations, apparently involves only a part, as the intellect, the emotions, or the will, or certain manifestations only of a faculty of the mind; which originates in the intellectual faculties rather than in the feelings; and the manifestations of which are well-defined, persistent, dominant, and systematic in character.*

## Clinical Cases.

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### REPORT OF A CASE OF PROGRESSIVE MUSCULAR ATROPHY WITH BULBAR SYMPTOMS; THE ATROPHY LIMITED TO THE LEFT UPPER EXTREMITY, THE ABDUCTOR INDICIS OF THE RIGHT HAND, AND THE TONGUE, WHICH IS AFFECTED BILATERALLY, PREDOMINANTLY ON THE LEFT SIDE.<sup>1</sup>

By W. R. BIRDSALL, M.D.

I would hardly be justified in presenting to a society devoted to neurology a single case having the usual features of progressive muscular atrophy. On the other hand, the study of such cases as present deviations from the common type, even if such deviation be not great, are frequently of value, if they serve no other purpose than that of exhibiting the non-conformity of disease to our arbitrary standards of classification and description of types of disease.

The patient before you shows peculiarities due to the pathological process having advanced to an extreme degree in certain regions, while its distribution has remained limited; not involving neighboring parts usually affected; the left upper extremity and the tongue being the parts invaded, and these to a marked degree, while the right upper extremity is normal, with the exception of the abductor indicis; the trunk and lower extremities remaining healthy.

His history is as follows A male, æt. 39, a native of Sweden, of occupation a carpenter, married, of temperate habits, and with no history of syphilis or other disease. One year ago, two weeks after his arrival in the United States, observed the first symptom of his present disease, namely, a difficulty in articulation from a slowness of certain movements of the tongue. A month later, weakness in the left thumb and forefinger became troublesome in holding a nail. Weakness of the biceps was observed next, and

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<sup>1</sup> Read before the New York Neurological Society, Dec. 7th, 1886.

by another month the entire left upper extremity, including the shoulder movements, became perceptibly paretic. A month later, atrophy of the small muscles of the left hand, chiefly the abductor indicis, was detected by the patient. Paresis and atrophy slowly increased, most rapidly in the tongue; deglutition became affected to a slight degree three months after the speech disturbances. Salivation also increased. Fibrillary contractions and the sensations which accompany them were observed by the patient in the upper and lower extremities and in the trunk, but no other sensory disturbances were noticed except that of a deep-seated, stabbing pain in the left biceps, not intense, and a sensation in the tongue which he describes as "a sleepy feeling." Taste has failed during the past year, particularly for delicate tests. He gives no history of other disturbances of the special senses. His mental, alimentary, urinary, and genital functions have remained normal.

**Family History.**—His father died with dropsy. His mother had some disturbances of speech, lasting for six months, between her fortieth and fiftieth years. She still lives and is eighty years old. He has five sisters and two brothers, all older than himself, who are all alive and without history of disease except one sister who died during childbirth.

The patient was first seen by me at Dr. Seguin's clinic for nervous diseases at the College of Physicians and Surgeons, and was referred to my service at Manhattan Eye and Ear Hospital for electrical treatment.

**Examination.**—Tests of vision and ocular mobility were kindly made for me by my friend, Dr. J. B. Emerson, at the Manhattan Eye and Ear Hospital.

R. V. Hyperopia  $\frac{1}{4}$  s. Accommodation  $\frac{1}{4}$ . No insufficiency.  
L. V. Hyperopia  $\frac{1}{8}$  s.

Pupil normal, iris reacts normally to both light and accommodation. Fundus normal.

Hearing normal.

Facial movements normal except orbicularis oris. Attempts to whistle are imperfect, sucking movements and kissing movements are also imperfect, the labial sounds in speech are weak.

The tongue presents a convoluted appearance of its mucous membrane, due to muscular atrophy, more marked on the left side. He cannot lift the tongue from its bed, nor touch the hard palate with the tip of the tongue, but can protrude it imperfectly and make slow lateral movements. Fibrillary contractions are present to a marked degree.

The left palatal arch is somewhat lower than the right. The throat reflexes are very active. The disturbances in deglutition appear to be due more to defective lingual movements than to abnormal pharyngeal action. The inequality in the palatal arches may possibly be due to the same cause, as it is only apparent in a passive state.

**Speech.**—The lingual sounds are the most defective, the labials less so. These defects are not so apparent in the utterance of in-

dividual sounds as in continuous speech, and particularly in such words as require sudden changes in the position of the tongue. His wife's name, for example, "Tilla," is difficult for him to articulate. The sound "Th" is quite defective and others which require the approximation of the tip of the tongue to the hard palate. While his speech is not strictly speaking syllabic, there is a slowness of articulation which makes syllabication more distinct than in normal speech; at the same time this slowness is not at all like the slow and slurring speech met with in cerebral cortical degeneration. He can hum tunes as correctly as ever, and his laryngeal functions are normally performed. He thinks, however, that he is not as strong as formerly in shouting in the open air. He has no shortness of breath. Can run as fast and as far as ever, but with more fatigue than formerly. There are no cardiac or pulmonary defects.

Motility, sensibility, and nutrition of the lower extremities are normal, except occasional slight fibrillary contractions. The patellar tendon reflex is active and equal on both sides. The cutaneous reflexes are normal.

*Upper Extremities.*—The muscles of the entire left upper extremity are of less volume and more flabby than those of the right side. In the left hand, of the abductor indicis but little muscular tissue remains; that which is present abducts the finger alone, but cannot overcome much additional resistance. The thenar group of muscles are flabby and diminished in volume; the dorsal interossei and abductor minimi digiti also show diminished volume and enfeebled power. The flexors and extensors in the forearm, though fairly well preserved, lack the rounded form and the hardness of the corresponding muscles on the right side. The biceps exhibits decided wasting flabbiness and feeble action. The deltoid, the pectorals, and the infraspinatus also show diminished power and volume, particularly the last-named muscle.

*The electrical reactions* are shown in detail in the accompanying chart.<sup>1</sup> They may be summarized as follows: There was diminished faradic excitability of all the affected muscles, as compared with those of the opposite side; this diminution being relatively greater on direct excitation than through the nerve, even the few remaining fibres of the left abductor indicis responding proportionately to its volume. Slowness of contraction was apparent, particularly in the small muscles of the hand, and to a less degree in the biceps. The abductor indicis was the only muscle of the right side which presented diminution and slowness of contraction. With a reversible electrode, difference in polar action was noticeable in both the right and left abductor indicis, consisting in a reversal of the normal formula of contraction, the anode producing a stronger contraction than the cathode.

With Galvanism.—Quantitatively diminished reactions were found in the affected muscles by direct excitation, while through

<sup>1</sup> Omitted in publication.



the nerves, excepting the left abductor indicis, the reactions were about equal to those of the opposite side when allowance was made for the difference in the volume of the muscles. Qualitative changes, consisting in slowness of contraction and a reversal, complete or partial, of the formula of contraction, were found in both the right and left abductor indicis, on the second interosseous and the ulnar distribution of the thenar group of the left side. The reaction on the left abductor indicis by excitation of the ulnar at the elbow, besides being quantitatively *increased*, gave the unusual result of an anodal closure contraction equal to or greater than the cathodal closure contraction.

The reactions of the biceps to galvanism are interesting, as showing the result of currents of different strength on the formula of contraction in a degenerating muscle.

With 6 M.A. the reaction (by direct excitation) was

K.C.C. > A.C.C.

With 8 M.A.

K.C.C. = A.C.C.

With 10 M.A. < A.C.C.

The anodal contraction was the one which increased in volume, the cathodal being nearly as great with 8 M.A. as with 10 M.A.

The muscles of both sides show increased excitability to mechanical excitation.

Sensibility.—While the patient has been under treatment at the hospital, both Dr. Cramer and myself found diminished sensibility to both the faradic and the galvanic current on the entire left upper extremity, but not elsewhere. At the last examination, December 2d, this could no longer be detected.

In testing sensation with the galvanic current, a considerable difference in resistance was observed between the two sides on the finger tips, which on measurement was found to be 1,000 ohms, showing the importance of working with a milliampère meter in making quantitative tests. If the number of cells required to produce like sensations on the two sides had been taken as a measure, the fallacious conclusion would have been reached that one side was more sensitive than the other.

In the tongue, quantitative diminution, slowness of contraction, and reversal of the formula of contraction were found on direct excitation with both the faradic and the galvanic current.

## A CASE OF INTERNAL HYDROCEPHALUS, DUE TO DISEASE (THROMBOTIC) IN THE WALL OF THE STRAIGHT SINUS.

BY WM. BROWNING, M.D., OF BROOKLYN.

The causes of internal hydrocephalus, exclusive of the forms due to intra-ventricular inflammation and compression of the venous discharge by tumors, are little known. A case which would ordinarily be called idiopathic, but in which a sufficient cause was found, is therefore worth recording. It occurred in a six-year-old girl of German parentage. The first symptoms of the trouble began three months previously; these it is only necessary to state briefly. She had been apparently well until attacked by vomiting. This was followed by convulsive attacks and later by a variety of indefinite symptoms; opisthotonus in the convulsive seizures, pain across the forehead or in one or the other ear, general weakness, etc. No paralysis, no trouble with vision, intelligence clear to the last.

She died under the charge of Dr. Bender, with whom I made the autopsy and to whom I am indebted for the above notes.

The skull was thick and firm. Brain surface and envelopes healthy except as to the sinuses. No trace of meningitis on either base or convexity. On removing the brain, clear fluid broke through the posterior perforated space. The whole amount of fluid was estimated to have been 5-6 oz. The superficial gyri were but very moderately flattened. Examination of the ventricles showed the velum interpositum firm and rather thick. No adhesion or other sign of inflammation in the ventricles. Both lateral chambers, and the third and fourth ventricles with the connecting iter were dilated. The only noticeable alteration in their walls was the dilatation of the veins, especially in their finer branches. This was apparent on the ventricular roof as well as floor (most of the roof-veins discharge through the vena Galeni). The two *venæ cerebræ internæ* were very broad and contained liquid blood. No cause for the trouble then was discovered in the distended cavities. On examining the sinus rectus, a dark thickened spot immediately attracted attention. This was about half an inch from the anterior end. Starting from the torcular a director readily passed up the sinus until this point was reached where an obstruction was met. On slitting up the sinus, it was found that opposite the thickening an oblique thin membranous

septum had retarded the sound. After opening, it was not possible to say whether the membrane had completely closed the narrowed sinus, the sound having made an opening, or whether a fine slit through it had existed. Immediately adjoining this were several fine fibres crossing from wall to wall, but not like the bands so often seen in the various sinuses. The above-mentioned thickening in the sinus wall affected each side and was in the substance of the wall itself. On cutting through either side, a layer of reddish-gray organized material was found just in the position of the parasinoidal spaces which occur, in adults at least, even along the straight sinus. This deposit extended about a third of an inch, the remainder of the sinus being free. The longitudinal sinus showed a somewhat similar though less advanced condition. Its main channel was everywhere free, but the *sini subalterni* shone through full and black and to the feel presented firm cords. On opening these, a dark fibrinous material, partly organized, was found firmly attached to the surroundings. Here evidently a process had been going on, quite similar to that beside the straight sinus, but of a more recent date. Moreover, lying below the vessel's channel these thromboses did not materially contract it.

The apex of the right lung was adherent and very hyperæmic, though not presenting any discoverable tubercles.

We know from many cases of cerebellar tumor that compression of the venous discharge from the ventricles may cause internal hydrocephalus. In this case the obstruction developed in the main efferent vessel itself, and hence was not quite parallel, since certain anastomoses may have remained free. As I have elsewhere shown ("*Veins of the Brain*," 1884), the ventricular veins are terminal vessels, their only connection with other veins—except by the sinus rectus—being in their posterior portion just before uniting. Here certain basilar veins, usually discharging through the vena Galeni, communicate in a roundabout way with other cerebral veins. This limited anastomosis was doubtless but slightly interfered with, except secondarily by the increased pressure from the accumulating fluid.

No history of syphilitic taint or other cause for the peculiar thrombotic condition could be found.

## REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILA- DELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF W. H. WALLACE M.D., PHYSICIAN-  
IN-CHIEF, AND CHARLES K. MILLS, M.D., CONSULTING PHYSICIAN.

CASE XII.—Delirium grave. (Typhomania, mania gravis, phrenitis, or acute delirium.)

Reported by Harriet Brooke, M.D., Assistant Physician.

E. K., female, aged 39 years, married; was admitted to the hospital Sept. 9th, 1886. She was an American, had a fairly good education, was a Methodist, and by occupation a maternity nurse. She was not addicted to the use of liquor. There was no history of epilepsy or insanity in her family, and none of any head injury. Her head was normal in its conformation.

The patient was brought into the hospital by several men; was raving violently and struggling, throwing her long thin arms about her in anaimless convulsive manner. According to her husband's account, this condition of excitement had come on within five days previous to her admission. She had a number of bruises about her person, chiefly located in the vicinity of the joints—as the knees and elbows—in such positions that they had probably been inflicted upon herself by the violence with which she had tossed herself about.

A physical examination was made with difficulty. Heart and lungs were found normal, and the abdominal organs were apparently healthy. Vaginal examination revealed a slight retroversion of the uterus.

Inquiry into her family history revealed the fact that her domestic life was an unhappy one; her husband drank heavily, and she had been on bad terms with her own family. Just before the attack came on, she had separated from her oldest son who was going on a journey.

A ten-grain calomel purge was given and a hypodermic injection of hyoscine hydrobromate, after which the patient slept two or three hours; but on awakening her excitement was very great. Her face, words, and gesticulations were weird and awful in the extreme. She would crouch in a corner and with a rhythmical to and fro movement of her head and one hand whisper, "death, death, death," in a most ghastly fashion, all the while looking

straight into the eyes of those about her with a piercing and terrified stare. Then in a distressed voice she would call "Jennie," after which she would declare one minute that she had murdered "Jennie," and the next that she must murder her. She then became possessed with the idea that the nurse was Jennie, and grasped her violently.

The following is a further record of the case:

Sept. 8th, 1886.—The patient was found in a condition of wild delirious excitement, the convulsive and rhythmical movements much increased and involving the whole body. There was marked retraction of the head at the same time that she tossed it from side to side. She had a small, rapid, and very feeble pulse; her temperature was taken with difficulty, and found to be 104 in the axilla; her eyelids were forcibly closed. It required the best efforts of four nurses to hold her and to restrain in some degree her violent movements.

The actual cautery was applied to the back of her neck and an ice cap to her head. Croton oil was administered internally, followed by a turpentine enema, as the bowels were obstinately constipated.

Her pulse was meanwhile rapidly growing weaker, and her condition becoming very like that which immediately precedes dissolution. She was given a hypodermic injection of ether, and in a few minutes one of digitalis and ergot, followed soon by another of ether. A large mustard plaster was applied over the heart, and before long her pulse responded to the remedies employed. The croton oil also operated effectually, and reaction was established.

The convulsive movements, though less, were still continued, and sleep seemed out of the question without a narcotic. A hypodermic injection of morphia was given, but with the result of increasing the delirium. Half an hour later a hypodermic injection of hyoscine hydrobromate was given and was followed, in a very few minutes, by a quiet sleep; but she soon lapsed again into a condition of wild excitement, not so great, however, as that which preceded the sleep. More hyoscine was then given and again she slept. The delirious excitement, retraction and tossing of the head, high temperature, rapid pulse, and sleeplessness, except when sleep was induced by hyoscine hydrobromate, continued for five days, during which time retention of urine and albuminuria were present. Her bowels were also obstinately constipated, and after the immediate effect of the croton oil had passed off were moved by enemata only.

One favorable feature in the case, however, was the fact that the woman had thus far swallowed mechanically and retained all the nourishment that was placed in her mouth. Advantage was taken of this, and she was given abundantly of eggs, milk, and beef extracts. Whenever her temperature rose to 104°, it

was reduced by the cold pack, which was resorted to several times during the progress of the disease.

The amount of hyoscine used to induce sleep was so great that it caused a very marked dryness of the throat and tongue. It was suggested that pilocarpine might correct the trouble, and one-quarter grain was given hypodermically, and in fifteen minutes more one-eighth grain, and this was followed in a very few minutes by a normal secretion of saliva, which moistened and softened the tongue effectually. The skin also became quite moist, but there was no profuse perspiration or salivation. Besides the hyoscine, which was given at night, the patient had potassium bromide, xxx. grains, every two hours throughout the day, during part of the time.

The following is a note taken on September 13th, 1886:

The patient is improving; retraction of the head is much less marked; raving and incoherent delirium have somewhat subsided, and the amount of albumin in the urine is diminished; morning temperature,  $103^{\circ}$ ; afternoon,  $99.4^{\circ}$ . Hyoscine always induces sleep and thus controls the delirium.

Sept. 15th, 1886.—Bromide and hyoscine are continued. The patient is not so well; she is raving again. It was noticed that while the hyoscine produced sleep and quieted the patient for a time, yet on awakening she soon lapsed again into a state of excitement. Whether this was a secondary effect of the drug or the natural course of the disease might be a question, but it is certain that the hyoscine had no influence in preventing it.

Sept. 17th, 1886.—Bromide and hyoscine were both discontinued, and paraldehyde, fl. 3 ij. given per rectum at night.

Sept. 18, 1886.—The patient slept very well indeed after the paraldehyde enema of last night, and did not waken till this morning; she seemed much quieter and better than at any time before; she talked rationally, and both motor and mental excitement had disappeared. Temperature  $98^{\circ}$ ; pulse  $86^{\circ}$ . She seemed very weak in the afternoon; she was quiet as in the morning; temperature  $98.6^{\circ}$ .

She objected to swallowing to-day, probably because her bromide was given to her in milk and she was afraid that all her food contained medicine. She was fed, therefore, by the nasal tube.

Up to this time she had had two hypodermic injections of hyoscine<sup>1</sup> through the night, one at 8.30 and the other when she awakened from the effects of the first, and became noisy and violent.

Sept. 23d, 1886.—The patient was rapidly convalescing; albumin has disappeared from the urine. Temperature and pulse are

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<sup>1</sup> The preparations of hydrobromate of hyoscine are not all of the same strength. The one used in this case, bought with the understanding that it was Merck's hyoscine, was given in doses of  $\frac{1}{4}$  grain. It was found not to be Merck's, and hence the large dose given. The usual dose of Merck's hyoscine is  $\frac{1}{60}$ ,  $\frac{1}{80}$  of a grain.

normal, and she has a remarkably good appetite. Her bowels are constipated; calomel and sodii bicarb. were given in divided doses.

The patient went out with her son on October 8th perfectly restored to health and reason. She came back again the latter part of November to see if she could not find employment in the house. She looked very well indeed, and the frightful mental storm through which she had so lately passed, seemed to have left no trace on her mind.

This, I believe, is a very unusual termination for delirium grave. Spitzka says of its termination: "The majority of patients afflicted with delirium grave die in the delirious period after an illness of a few weeks; the excitement continues unabated for four or five weeks. The subsequent symptoms of stupor increase and the history closes with a fatal coma. Complete recovery never occurs; in rare instances, the patients emerge from this severe disorder with a slight defect; in others, paretic and terminal dementia supervene."

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## Correspondence.

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MARCH 16TH, 1887.

*To the Editor of the Journal of Nervous and Mental Diseases.*

SIR:—Since the publication of my article on "Recoveries from Insanity in cases accompanied by Hæmatoma Auris," in the February number of your JOURNAL, in which I stated that "these four cases comprise, I believe, all that have thus far been reported," my attention has been called by Dr. Wm. Noyes, Assistant Physician at the Bloomingdale Asylum, to two similar cases that were reported by Dr. Brown, of that institution, now in Europe, in the *Medical Record* of June 19th, 1886.

CARLOS F. MACDONALD.

State Asylum for Insane Criminals,  
AUBURN, N. Y.

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## Society Proceedings.

### NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, February 1st, 1887.*

*The President, C. L. DANA, M.D., in the Chair.*

DR. C. L. DANA reported a case of

#### PACHYMENINGITIS HÆMORRHAGICA,

with large meningeal hemorrhage pressing chiefly on leg centre ; right hemiplegia ; total paralysis in leg : aphasia ; hemianæsthesia ; convulsions limited to arm and facē ; death.—Exhibition of specimen.

The patient was a woman about 68 years old, and came into the hospital with complete motor aphasia, and unable to give any previous history. She had no paralysis at first, but three days after admission she had a general convulsion, followed by right hemiplegia, total in the leg, and some right-sided anæsthesia. On the second and third days, she had a series of brief localized convulsions, involving the face bilaterally and the right arm. These were carefully observed. The movements were clonic, beginning in the muscles of the lower jaw. The other peculiarities were these : 1. The pupils remained small during the convulsions. When wider convulsive centres are discharging, as in general epileptic convulsions, the pupils are dilated. It is not probable that in this case there was some uræmic element, because the post-mortem disclosed a sufficient cause for them. 2. The conjugate deviation of the eyes was at first, and very temporarily, toward the side of the lesion, and away from the paralyzed side. The head also was turned toward this side. When this occurs, it is ordinarily spoken of as a paralytic deviation. This does not explain it here, since almost immediately the head and eyes were turned strongly to the opposite and paralyzed side.

The speaker suggested that the first deviation is due to an inhibition of the activity of the associated nuclei of the third and sixth nerves that innervate the external and internal recti of the two



eyes. There are many facts which tend to show that the first stage of convulsion is a transient paralysis due to a sudden discharge of inhibition centres. These are of a higher, more developed class than the centres for motor discharges, and would be affected first. We would have then loss of consciousness, inhibition of motion, and muscular relaxation; then motor discharges and tonic and clonic convulsions.

3. The temperature on the paralyzed side was one degree higher than normal, and higher by a degree than that of the other side. This is the rule in intra-cerebral hemorrhage and hemiplegia, but the speaker was not aware that it has been established in cortical hemiplegias. In meningeal hemorrhages, the temperature is often below normal, according to Minot. 4. The presence of hemi-anæsthesia.

The patient died on the third day. Post-mortem showed chronic pachymeningitis over both convexities, but more on the left side. On the left convexity there was a very extensive fresh meningeal clot pressing upon and flattening especially the upper half of the central convolutions. Brain substance normal.

CORTICAL EPILEPSY WITH TEMPORARY APHASIA. SYPHILITIC GUMMA COMPRESSING THE LEFT SECOND FRONTAL CONVOLUTION IN ITS LOWER POSTERIOR PART. RECOVERY.

DR. M. ALLEN STARR related the history of the case. Chas. S., aged 32, had always been healthy and a hard worker. He had an attack of sciatica four years ago, and three years ago had a hard chancre. He had never had convulsions or nervous affection. Family history good. During November, 1886, and the first two weeks of December he did not sleep well, was slightly dizzy, his head ached a good deal, chiefly at night. December 15th, while walking with a pail in his right hand, he suddenly let it drop, losing all power in the hand and arm. There was numbness in the hand. He was unable to speak to his companions. He did not feel dizzy or notice any pain in the head; he did not lose consciousness nor fall. He understood his friends' questions, but could not answer. Power in the hand and arm and speech returned within half an hour. The next morning he went to work as well as usual. Two days later a second attack occurred, beginning with a numb feeling in the tips of the fingers, gradually extending up the hand and arm. Then the fingers became rather forcibly flexed and stiff, but by a voluntary effort he could

straighten them. No clonic spasms of the fingers, and wrist and elbow were not bent. The numbness and stiff feeling soon extended to the face, which was drawn to the right side with some force. Speech was again lost. No loss of consciousness. The attack lasted about twenty minutes. Such attacks had occurred every other day, then every day, and finally twice a day up to January 3d, and during this time the headache and insomnia were increasing steadily. The character of the attacks was not uniform. Sometimes the spasm would begin in the face, though usually the arm was first affected. Both were involved in every attack, but the spasm and numbness never reached the leg. The hand felt cold during the attack, though warm to the touch. On one occasion he had for four days great difficulty in making himself understood by words. Examination by Dr. Starr showed slight paresis, and slight tactile anæsthesia in right hand. No affection of face or speech. No cardiac symptoms, though suffering from headache. Percussion of skull did not reveal any tender spots. Thrombosis, endarteritis syphilitica, diffuse encephalitis with sclerosis were excluded, and the diagnosis was reached of gumma in the membranes, resting upon the brain surface, giving rise to irritation and consequently to an occasional nervous discharge, but not of sufficient size to cause any destruction. Location of tumor was equally clear, the relative situations of the cortical centres for the arm, face, and for the movements of speech in the lower two-thirds of the anterior central convolution and in the posterior part of the third frontal convolutions were likened to a reversed L. All these centres were irritated during the attacks, the irritation sometimes beginning in one, sometimes in another. If the tumor pressed upon the lowest posterior part of the second frontal convolution which would lie inside of the L, an irritation radiating from it might reach all three centres equally. The total intermission of the local symptoms might be explained by such a location, since no symptoms were known to occur from injury of this part. The fact that numbness in the hand and face uniformly accompanied the attacks of spasm seemed to indicate that the areas for these parts coincide with the motor areas.

Another point of interest was the distinctly motor character of the aphasia.

The treatment ordered was first inunction of mercury, and secondly iodide of potash daily in divided doses. He had one attack two days after beginning treatment, but since that time he had

had no return of the symptoms. Iodide of potash was still being taken.

DISCUSSION ON DR. STARR'S PAPER.

DR. SEGUIN had seen several cases whose symptoms resembled those of the case recorded in Dr. Starr's paper. The prognosis of even non-syphilitic cortical lesions with this symptomatology was not absolutely unfavorable. One of the cases to which he referred was that of a Cuban who came to his clinic about nine years ago. He had never had syphilis, yet he described epileptic attacks of the true cortical kind, such as have been obtained by experiments upon animals during the last few years. The hand would become numb, and then the seat of a vibratory sensation; finally contraction would occur in the hand, when the face, and almost simultaneously the leg would be affected, and he would lose consciousness. According to his friends' account, general convulsions then occurred. He had had quite a number of these seizures, yet examination showed no anæsthesia, no affection of the optic nerve, and, so far as the speaker could recollect, no motor impairment. The patient had received a preparation composed mostly of the bromide of potassium, to which a little of the iodide was added. He improved immediately, and four years ago the attacks ceased. Once in a while he has the sensation of wires in the hand, and the hand becomes stiff, but the face is never affected. The case was a beautiful illustration of the localization of a lesion in the centres for the hand, the discharge radiating to those of the face and leg of the same side, then to the opposite side, with loss of consciousness.

The patient had also been the object of the bracelet experiment. He was a powerful man, and had exerted great force, arresting many attacks in this way. The speaker was satisfied that syphilis was absent, while the amount of the iodide was too small to explain a cure upon the ground of a syphilitic affection.

DR. STARR had been much interested in the case which Dr. Seguin had related. He had recently had a case of unilateral convulsions in his office. The patient was a small boy. The attack commenced in the eyes and face. The eyes turned to the right, then the head turned to the right, then the arm, then the leg became affected. During the attack, the speaker had asked suddenly, "What is your name?" The boy promptly replied, "Arthur," and then relapsed into the convulsion. He supposed that the reply was reflex, as the boy was unconscious at the time and did not

afterward remember the occurrence. He would like the opinion of the members upon the point.

DR. DANA asked what Dr. Seguin had considered the lesion in his case.

DR. SEGUIN had never ventured to surmise beyond the fact that there was a nerve lesion, and that there was no syphilis in the case.

DR. STARR asked Dr. Seguin whether in localized convulsions numbness were not the rule.

DR. SEGUIN replied that it was, but he did not know that the reason was yet sufficiently established, although Von Monakow had associated anæsthesia with lesions of the motor zone.

DR. SHAW referred to a case seen first four years ago. While at work as a jeweler, the patient fell off his bench in a convulsion. The face and the left arm were convulsed; the leg was not affected. Sometimes only the side of the face was affected. He had seen many of these attacks limited to the side of the face in his office. The patient complained of numbness in the arm and the side of the face, and the speaker felt sure that the tactile sensibility was not as good upon that as upon the other side. The patient denied syphilis. Upon ophthalmoscopic examination the nerves were found pale, and the visual field restricted in its upper part. There was no change until about six months ago when, without the loss of vision, he was found to have choked disk. This had gone on to atrophy, and the man was now blind. There was no paralysis. From the choked disk, of course, the speaker had now diagnosed a tumor, but he referred to the case on account of the anæsthesia and the spasms, and their resemblance to those in Dr. Starr's case.

DR. SACHS referred to the case of a man who some years ago while working upon the Capitol at Albany, had fallen some distance, was found unconscious, but recovered. A few weeks later he developed symptoms which alarmed his friends, and he had now some of the physical and nearly all of the mental signs of general paresis: the irregular pupils, the facial tremor, the tremor of the tongue, and the deteriorated mentality. The speaker referred to the case because of the traumatic incident, and because every three or four weeks this man had an attack of numbness beginning in the fingers and creeping up the right arm to the face. There never were convulsions, but both the patient and his wife, who is a very intelligent person, claim that paralysis follows

upon these attacks of numbness. After three or four hours, both the paresis and the numbness disappear, and he has a very severe headache, lasting one or two days. The speaker thought there was a question of chronic meningitis with encephalitis possibly in this case. It was evidently a cortical affair.

DR. DANA thought that cortical epilepsy might develop, like idiopathic epilepsy, without an appreciable lesion. He recalled a case, that of a young man who was kicked in the front of the thigh by a horse. Twitching of the leg developed, similar to that of cortical epilepsy. Thrilling and numbness of the arm and face followed. In a year true hemiepileptic attacks, during which he lost consciousness, developed, and upon giving him ether for stretching the nerve, he went into the status epilepticus. There was no history of syphilis. Apparently cortical epilepsy was developed just as true idiopathic epilepsy in other cases.

DR. E. C. SEGUIN read a paper entitled,<sup>1</sup>

A CONTRIBUTION TO THE PATHOLOGY OF THE CEREBELLUM.

DISCUSSION OF DR. SEGUIN'S PAPER.

DR. BRADNER spoke by invitation. He had been the attendant in the case of the child to which Dr. Seguin had referred. He had not prescribed the washing out. It had been done by a prominent physician of the place. He had done it a number of times during three years, but had then refused, believing that the child had brain disease of some form. He saw the patient first in November. The vomiting was always in the morning. There was no pain connected with it then, although a frontal headache had developed during the last few months. The treatment prescribed by Dr. Seguin had been the iodide of potash fifteen grains t. i. d., increasing five grains daily until 100 grains were taken at a dose. The course had been interrupted by several attacks of acute gastritis, but the child had had those attacks previously—they did not appear to depend upon the medication. The eyesight has been perfect; the child could detect the smallest point made by a lead pencil or needle. While using the iodide his headache had improved, as had some other symptoms, but he retained his old man's gait.

DR. SEGUIN remarked that, though seeing well, the child had typical choked disk.

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<sup>1</sup> See this JOURNAL, p. 217.

DR. BRADNER added that since seeing Dr. Seguin he had obtained a history of injury in the case. Two years ago, and just before the commencement of his illness, he had, while trying to skate, fallen and received a severe blow on the back of his head. One result of this injury had been abiding terror at the sight of a body of water or ice.

DR. SHAW had shown a child at the American Neurological Association in 1878, on account of a peculiar ataxic gait like that of locomotor ataxia. The later symptoms had pointed to a tumor of the cerebellum.

DR. PUTNAM JACOB referred to the recent collection of cases by Bernhardt. All cases published previous to 1878 had been collected by Nothnagel. Dr. Seguin's cases tended to confirm Nothnagel's laws. There were many resemblances between the symptoms of cerebellar tumor and those of tumor in other parts of the brain. The peculiar violence of the headache and the choked disk found in most cases of cerebellar disease might, she thought, be due to the increased pressure of a tumor confined by the tentorium. This element had not been commented upon, and she would like Dr. Seguin's opinion upon it.

Another point referred to the fact that the laws formulated by Nothnagel recognize the possibility of complete latency of the tumor, no symptoms at all being present when but one lobe of the cerebellum is affected; such symptoms appearing only when the tumor encroaches upon the central lobe. In one of Dr. Seguin's cases the tumor only occupied the lateral lobe. Of course, an indirect affection of the central lobe might be present even in such a case.

DR. LESZINSKY referred to a case to which he was called in consultation by Dr. Alexander. The boy had the typical gait of spastic paraplegia; any attempt to stand caused spastic contractions in the limbs. The ankle clonus was present, and the knee-jerk was exaggerated. There were no cerebral symptoms; the fundus was normal in both eyes. The father and mother were both alcoholic and unworthy people. Finally, the child became unable to walk, but there were still no cerebral signs, no vomiting. Two months before death the fundus was still normal. Later, tremor developed, and paralysis of the abducens, of the fifth, and of the third nerves. Total blindness occurred. The nurse, a graduate of Bellevue, was positive that the child could see nothing. It died in a convulsion. Unfortunately, the body was immediately

frozen, and the specimen was unfit for sections. The tumor was found occupying one side of the cerebellum. The spinal cord was not fit for examination. There was a well-developed meningitis, and the paralysis was accounted for by considerable exudation about the cerebral nerves. One peculiarity had been a subnormal temperature during one stage of the case.

DR. STARR had had occasion some time ago to make a collection<sup>n</sup> of cases of cortical lesions from American literature. He had at the same time made a collection of cases of cerebellar disease from the same sources. As the data obtained only corroborated Nothnagel's results, he never published them. But Dr. Seguin having remarked upon the value of corroborative evidence, he would briefly refer to them now. From 1860 to 1884, one hundred and sixty cases of cerebellar disease were reported in American literature. In only 40 of these were the symptoms and the autopsies described with sufficient accuracy to warrant conclusions. These 40 the speaker had quite thoroughly analyzed. In 4 there were no symptoms; in 1 of these there was congenital atrophy of the cerebellum; in 2 abscesses; and in 1 a large cyst. Of the remaining 36 presenting symptoms, there was headache in 36; inco-ordination in 25; vertigo in 20; vomiting in 18; blindness in 14; dim vision in 6; diplopia or strabismus in 7; deafness in 7; facial spasm or paresis in 4; hemiplegia in 9; general paralysis in 4; mental symptoms in 8; stupid 7; mania 1; convulsions in 7; sexual desire increased in 2. Males 23, females 17. Ages, between 1 and 20 years, 11 cases; between 20 and 40 years, 16 cases; between 40 and 60 years, 9 cases; over 60 years, 1 case; age not stated in 3.

In two of the eleven cases in which inco-ordination did not occur, the lesion probably involved the middle lobe. In cases where inco-ordination occurred, various parts of the cerebellum were involved, but the probability was that the middle lobe was affected in the majority. There were only two instances of increased sexual desire. Bernhardt had found but one instance in ninety cases of cerebellar tumor, and Nothnagel but two cases. The speaker thought that it might be thrown out as a symptom of cerebellar disease, and regarded as one of accidental occurrence. The escape from vertigo in Dr. Seguin's case was explained, perhaps, by the recent discoveries in the anatomy of the course of the acoustic nerve. This nerve served for the sense of hearing and the sense of space. The centre for hearing is in the pons. Edinger finds the centre for equilibrium in the cerebellum, to which acoustic

fibres pass by way of the middle peduncle. From this centre the central tract probably passes onward to the superior peduncle. Dr. Seguin says that the superior peduncle escaped in his case. It is therefore natural that vertigo should have not occurred.

The tendency to rotation was an interesting feature in these cases. There was a tendency to fall or turn forward in two cases: the lesion in both was a tumor in the vermis, in the anterior part. There was a tendency to fall to the right in two cases, in one there being a tumor in the left middle peduncle, and in the other an abscess in the same part. There was a tendency to fall to the left in two cases, in one there being an abscess in the right middle peduncle, and in the other a tumor in the left middle peduncle. A patient of Nothnagel, whom the speaker had seen in Vienna, when getting up in bed had always a tendency to turn to the right side. Nothnagel considered this due to vertigo. It was only present when the patient was erect. The patient felt as though about to fall to the left side, and hence turned to the right. This case had a tubercle of the left middle peduncle of the cerebellum. Nothnagel considered this symptom only produced by affection of the middle peduncle. In the two cases in the table where the tendency was to turn to the right, there was disease of the left middle peduncle; while in the two in which the tendency was to the left, in one the right peduncle, and in the other the left peduncle was affected. No rule can, therefore, as yet be laid down as to the cause of this symptom.

DR. SEGUIN closed the discussion. He was not surprised to learn of the little boy's fall, as he was a firm believer in the traumatic origin of these conditions. It was very difficult to obtain a history of fall. He could not give an opinion upon the question propounded by Dr. Putnam Jacobi. In regard to ascribing the vertigo to the acoustic nerve, he was not yet certain that the acoustic nerve had cerebellar origin. He thought it would be difficult to trace fibres through the lateral peduncle of the cerebellum, the vermis, and the anterior peduncle.

He said Dr. Starr had probably made a mistake when he referred to Nothnagel connecting rotation with disease of the middle peduncle. Middle vermis he had probably meant to say.

DR. STARR was aware that Nothnagel connected incoördination with disease of the vermis, but rotation towards one side was considered by Nothnagel as a symptom of disease in one or the other middle peduncle passing from the pons to the cerebellum.



## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, December 27th, 1886.*

*The President, DR. S. WEIR MITCHELL, in the Chair.*

DR. GUY HINSDALE read a paper entitled

FACIAL SPASM AND TIC DOULOUREUX FOR WHICH NEURECTOMY  
AND NEURO-TENSION WERE EMPLOYED.

Patient is 62 years old, was born in New Jersey, and has always lived there. She considers her home dry and healthy and free from malaria. Her father died with phthisis. The patient herself has never had malaria or chills and fever, but had an attack of pleurisy fifteen years ago, associated with a severe cough. This has been her only severe illness.

Twenty years ago the patient began to have twinges of pain in the lower jaw. Four or five years ago she suffered from attacks of neuralgia of the right side of the face two or three times a year: these attacks lasted from one to three months. In 1884 she had four or five attacks, one of which lasted two months; though quite weak, she was not confined to her bed at any time. Fever sometimes accompanied the attacks, without any chill, but with a cold sweat when the pain was very severe.

A year or two ago the pain occurred at any time during the day, but passed away at evening, upon retiring and remaining quiet. In April, 1885, the pain began to be worse at night, but it cannot be said that any special hour was observed. Cold air, eating, or drinking very hot drinks, motion in bed, bad news, mental trouble, aggravated the pain. Rest, peppermint-essence dropped on the tongue, sweating the face with hot bricks dampened with whiskey and wrapped in cloth, did for a time relieve the pain.

The treatment adopted consisted of rest in bed, good feeding, and the use of an ascending galvanic current. Cod-liver oil, 3 iv., and tinct. aconiti rad. were given after meals. The aconite was gradually increased in quantity until, on the nineteenth day, she was taking seventeen drops four times a day. Elixir of iron, quinine,

and strychnia, one teaspoonful thrice daily, was then added. The pulse varied in frequency from 67 to 82 per minute. Hydrochlorate of cocaine in four-per-cent solution was applied two or three times a day to the painful spot on the right side of the extremity of the tongue; this gave relief for about twenty or thirty minutes. Failing to control the pain notwithstanding this persistent use of aconite, on December 1st fluid extract of gelsemium was used; four drops were given thrice daily, and the dose gradually increased until the physiological effect was produced. Hunyadi water was given occasionally, and massage of the spine was adopted. On the following morning a blister was applied to the right temporal and zygomatic region; a cold-water dressing containing half a grain of morphine was used. The blister had caused considerable local irritation by evening, and the pain in the face and tongue was relieved; the pain, however, recurred the next day, with dull aching in the forehead and across the eyes.

Lunar caustic was applied to the right side of the tip of the tongue on December 4th; the area cauterized was about half an inch square. Electricity was suspended for three days. Subsequently applications of nitric acid were made to the tongue.

Swimming of the forehead, dimness of vision, and drowsiness accompanied the increasing doses of gelsemium. The dose of gelsemium was increased for twenty days, until the patient took thirty-six drops of the fluid extract three times a day; there were then dizziness, headache, some drooping of the eyelids, but no pronounced symptoms or apparent benefit from the medication.

At the end of six months the patient was still suffering with neuralgia, but not so severely as at first.

Mrs. E. H. H., æt. 64. Thirty years ago she began to have pain in the infra-orbital, and then in the supra-orbital region, darting and of intermittent character. It was serious enough to confine her to bed. The attacks were influenced by weather and exposure. Sometimes attacks came on every few days; sometimes not once in three or four months. Warmth would relieve the pain. Lini-ments failed; warmth always gave the most relief. Duration of attacks one to seven days, or even a month. For seventeen years she had noticed a singular sensation in the right nostril, as though pierced by a dozen needles. Pain extended to the cheek and under the eye. The patient for four years had a severe attack, lasting the whole month of February. It would take an entire month to recover from its effects.

The usual remedies proving of little avail, the infra-orbital nerve was excised by Drs. Hunt and Morton, a small portion of the floor of the orbit being also removed. The patient suffered a good deal of pain in the same spot as before; this, however, subsided to a certain extent, and two weeks after the operation she returned to her home in Maryland. While not altogether free from neuralgic pain, she considered herself much more comfortable than before and greatly improved in general health. Nine months later, she was reported to be in good health, with little or no neuralgia.

In a case of painless spasm of the face, Dr. W. W. Keen has recently operated on patient at the request of Dr. Wharton Sinkler. The patient had twice been paralyzed. Five years ago the right eyelids began to twitch, and in six months the whole face and the platysma were incessantly in spasm, which was increased by mental or muscular effort; later this was accompanied with constant pain. In June, 1884, the right infra-orbital nerve had been resected, with partial relief for only six weeks. Not long after this the twitching extended to the right side of the body and to the leg.

April 2d, 1886, Dr. Keen laid bare the trunk of the nerve, using an electric current to facilitate its discovery. The nerve was then stretched; the force used was just short of enough to lift the head. Total paralysis of the face followed, with relief not only of the spasm of the face and neck, but also of that of the side and leg.

Stretching of the facial nerve for cramp had been performed by Baum and Schüssler, in 1878, Eulenburg, Godlee, Hahn, and others.

Dr. Zesa's summary of nineteen cases gave permanent good results in three, doubtful in two, considerable improvement in four, failure in ten. In ten cases, temporary benefit followed the operation.

#### DISCUSSION.

The President, DR. S. WEIR MITCHELL.—I have several points to make with reference to these cases. The woman operated on by Dr. Keen I have seen within the past few days, and she presents no paresis of the face at all. As yet the case is a perfect success. She is entirely free from any twitching. Her case recalled others, and in looking over my note-books I found that within the past twelve years I had seen fifteen cases of painless tic, in none of

which was the slightest relief afforded by any method of treatment. It so happened that shortly after Dr. Sinkler's case was treated so successfully, Mrs. G., aged 49, the widow of a physician, a very intelligent woman, consulted me. She stated that her tic had begun about three years ago. It first appeared under the eye, where it nearly always begins, although I may say that as yet the history of these cases is not complete. Then the twitching appeared in the cheek, which, as a rule, has been the point next attacked in my cases. The spasm afterwards extended to the mouth. In those cases in which it became general, it extended beyond that; and in the last case that I have seen, the levator muscles of the ear were involved, so that the ear was drawn upward every time that the face twitched. But extension to the neck or more distant parts is seen at times in rare cases.

I observed in this case, as in some others, a certain amount of appearance of paresis on the affected side of the face, that is to say, the muscles on the sound side of the face appeared to predominate.

In this case, I began to think whether there was not something which had not been done which might give relief. The idea of weakening the facial nerve or its branches by freezing suggested itself. If I had reflected a little, I should have recognized the fact that it is impossible to affect the deeper nerves in this way. Freezing by vapor-jets applied to the surface acts very little beneath the skin. I have tried this by putting the finger in the mouth, and trying to freeze through the cheek by means of the rhigolene spray. I have never succeeded in producing more than a slight chilling. In this case, I have on sixteen occasions frozen with the rhigolene-jet a large portion of the skin of the cheek, sometimes covering the whole of the side of the face, taking care to protect the eyes and to prevent the inhalation of the vapor.

The result has been an interesting and a curious one. The very first freezing, like all the subsequent ones, brought on instantaneous spasm. The moment the jet struck the face, there was violent twitching. When freezing was effected, the spasms ceased and did not return for many hours. This result surprised me very much, and it appeared that while I was attempting to accomplish the result in one way, I had done it in another.

I observed that a vigorous freeze over the points of nerve-exit was better than a general freeze. I am about to try if localized freezing by ice and salt or other means which act more deeply

may not answer. The influence *seems* to be exerted through the sensory nerves, from which possibly arise also the impressions which create these spasms. With my remembrance of former cases, I cannot say that I feel hopeful as to the remote future of the present one.

I have tried the same plan of treatment in a second case of nine years' standing. She came to me some years ago, but, as I knew of nothing that would afford relief, I refused to treat her. I sent for her ten days ago, and began the use of the rhigolene spray. She, too, has slight paresis. The face is apparently pulled to the right, the spasm being on the left side, which is the side commonly affected. The results obtained with the individual freezings are practically the same as in the first case. The touch of the spray brings on the spasm. When the part is frozen, the spasms remain absent for a considerable length of time, although not so long as in the first case. The period varies from half an hour to two hours. Under this plan of treatment the attacks have diminished in frequency and in intensity.

After the spray has been used for some days, care must be exercised that a slough is not produced. I have never had a slough, although I have made the skin very sore. This has led me to think that possibly the good result was due to counter-irritation, and I shall not be satisfied until I have tried extensive counter-irritation over the origin of the facial nerve in some suitable case.

DR. J. MADISON TAYLOR.—I have had under my care a case in which a severe spasm of the right side of the face had been present for twenty years. The woman was nervous, and came of a family many members of which were in a general way nervous, yet quite strong and robust. The spasm, although very annoying, was not sufficiently marked to call for special treatment. About fourteen months ago the youngest daughter of this woman passed through a severe attack of typhoid fever. The mother, from nursing the child, became very much exhausted. Then the spasm grew worse, and was at times sufficient to shake the head. When the child became convalescent the mother was taken with the disease and had a severe attack, with high fever and delirium for six days. Upon her recovery from the typhoid fever the spasm had entirely disappeared, and to the present time, a period of ten or twelve months, there has been no return. It is interesting to note the curative effect of a severe illness on this intractable disorder. Hers was a veritable purgation with fire.

(To be continued.)

## Editorial Notes and Miscellany.

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WE learn that a bill for the Better Provision of Insane Criminals is now before the Committee on Ways and Means of the Assembly at Albany.

While considerable progress has been made toward the more successful treatment of our ordinary lunatics in State asylums and private institutions, the criminal insane (and how many of them are criminal because of their insanity?) have not been properly provided for in this or any other State, as far as we know. The asylum at Auburn has done pioneer work in this direction, but the report prepared by Drs. Stephen Smith and C. F. Macdonald, and State Comptroller A. C. Chapin has convinced us that that institution is sorely in need of improved opportunities and better facilities in the way of larger structures and available farm-land. At present, the proximity of the institution to the prison is decidedly obnoxious, while the lack of proper accommodations and the enforced confinement of a large number of insane within asylum walls deprive them of healthful exercise, and remove the one chance of recovery which work in the open air so often brings to these unfortunates. We hope, therefore, that the Legislature will grant the relief asked for, and that it will, above all, understand the necessity of supplying opportunity for agricultural work. The superintendent of the asylum at Auburn is fortunately on the committee; under his guidance and the watchful eye of Dr. Stephen Smith and Comptroller Chapin, we may rest assured that all plans will be ably and conscientiously executed.

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The Council of the American Neurological Association announces that the adjourned meeting of 1886 and the thirteenth annual meeting of the Association will be held conjointly at Long Branch, New Jersey, on Wednesday, Thursday, and Friday, the 20th, 21st, and 22d of July, 1887. There will be two sessions daily, one in the morning at 10.30 A.M., the other in the afternoon at 3 P.M.

THE  
Journal  
OF  
Nervous and Mental Disease.

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Original Articles.

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SIMULATED AND TRUE INSANITY IN THE  
CRIME CLASS.<sup>1</sup>

BY DR. WM. DUFFIELD ROBINSON,

PHYSICIAN TO THE EASTERN STATE PENITENTIARY OF PENNSYLVANIA.

I FIRST make the following explanation that you may correctly understand what is meant by the *Crime Class*.

Among the general populace there is a distinctly divided class, known by that descriptive title. In these the casual observer would see nothing characteristic. One experienced in penology can recognize a member of this class as easily as the native can an alien or foreigner.

The major part of state prison offences are the acts of men born members of this class of the people. They are of a kind peculiar to themselves. They are one of the morbid excrescences from society, and in progeny reproduce their peculiar kind. As the cancer cell lodged in a neighboring gland develops a growth identical in make-up with its paternal source, so the offspring of the crime class belie not their parentage.

These people very seldom reform, but in death end lives passed in crime and infamy. They have absolutely as

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<sup>1</sup>Read before the Philadelphia Neurological Society, March 28th, 1887.

little control over their natural inclinations to wrong-doing as the confirmed periodical drunkard has over desisting from his spree. They are very seldom given to rum drinking and rarely permit themselves to become intoxicated.

The proverbial "honor among thieves" occupies little, if any, place in their composition. They really see very little moral wrong in their crimes—the crime with them being in detection. They truly believe that the man who assists in their detection and aids in their being brought under the law is much worse than they themselves.

They do not believe in the existence of sexual purity and are nearly all given to excessive, promiscuous venery. This applies to both sexes. While often in possession of large sums of money, it is very speedily squandered, so that they are nearly always without financial resources. They have little regard for their lives and usually die without fear. Many are infidels or nearly so. They form strong attachments, but usually of an easily transferable sort. They have representation from all grades of intelligence and education.

Crime is *unquestionably* a monomaniacal infatuation with them. Statistics, attested facts, and direct attainable evidence clearly warrants this assertion. It is not an unusual case to have them acknowledge that they prefer the excitement of getting one dollar unlawfully to earning ten honestly.

The peculiar irregular development of intelligence in them is a surprising study. Some are exceedingly illiterate and ignorant, but possess a cunning and adroitness at deception that would compliment the wiliest fox. This peculiar cunning has been studied by many observers. It is either the result of direct inheritance, or the outgrowth of a peculiarly studied deceit practised from childhood in avoiding merited punishment. Their vital force, formative of intelligence, seems to be most strongly directed to this development. Each has some special line of criminal work, and to the repetition of it he adheres through life.

Physically they are a bad job, but, like bogus buildings, for a time would pass well on slight inspection. Their



cranium and intracranial contents are characteristic. Twenty-per cent at the age of 40 years have had syphilis. The worst component is their mental condition. Neurotic diseases are very common among them—epilepsy frequently exists; the different forms of mental diseases are developed in proportion far exceeding that common to the general inhabitants.

The study of the inherited tendency to mental and physical disease reveals much of valuable interest to the psychologist, the neurologist, and penologist. Fully ten per cent of them develop some sort of mental disease before death. Statistics kept over a prolonged period in the Eastern State Penitentiary show that of convicts who have been sentenced there three or more times (crime class), there have occurred in their immediate families—among father, mother, brother, or sisters—a startling proportion of deaths from tubercular diseases.

Three or more deaths from this disease occurred in the families of  $74\frac{1}{5}$  per cent of such convicts.

If the criminal, mental, and physical histories of the antecedents of the true crime-class man can be learned, it is found in almost every case that there has existed sufficient cause for him to have inherited the impelling criminal tendency of which he is possessor, and over which he has imperfect, if any, control. The want of control over this inherent force to crime is well exemplified in the following case:

A convict escaped from Sing Sing with ten years of a sentence yet to complete. He had over \$1,300 in money with him; he went into a small gentlemen's furnishing store to make a purchase, and unable to resist the temptation to steal, he purloined a necktie, instead of paying for it—thus jeopardizing his liberty to gratify a morbid propensity. He was detected, arrested, recognized by the police authorities, and with a sentence added, sent back to complete his former sentence. He knew the risk he ran, but could not control this propensity.

The perfect sanity of any of the crime class is disputed by some observers. Such a question involves their respon-

sibility, punishment, etc. The following, copied from our medical statistical register, shows the history of some recently received convicts, and also shows a relationship between the bad mental condition of their ancestors and the criminal lives of these their progeny.

A—Mother insane, father consumptive.

B—Uncle cut his throat while insane.

C—Insanity and consumption in family.

D—Father has been in insane asylum more than two years.

E—Epilepsy and insanity in family.

F—Epilepsy and insanity in family.

G—Six times in prison, mother insane, father consumptive.

H—Sister epileptic, mother weak-minded.

I—Insanity and consumption in family.

J—Impaired health, twice here; insanity and consumption in family.

K—Mother insane.

L—Father insane, mother weak and silly.

M—Sister insane.

N—Father insane for years, mother has periodic insanity.

O—Insanity in family.

P—Sister insane.

Q—Youngest of six brothers all in prison; his father was poisoned by his mother who had periodic insanity. Both grandmothers were thieves, sister weak-minded, uncle crazy. He had been in prison five times for burglary, three times for rape, and four times for horse stealing.

R—Mother insane.

S—Both parents insane.

T—Brother insane.

U—Mother insane.

About six per cent of the convict population shows a strong inherited tendency to insanity exhibited in their family histories.

That crime is often a family trait, by inheritance, is evidenced by the large number of convicts who have had

members of their nearest blood relations convicts, or who are so now. This is demonstrated by the following facts taken from the records of recent arrivals in our prison:

- 66 have brothers convicts.
- 9 have two brothers convicts.
- 4 have three brothers convicts.
- 2 have five brothers convicts.
- 12 have a convict father.
- 15 have convict uncles.
- 5 father and uncle convicts.
- 7 have sons convicts.
- 5 have a father and brother convicts.
- 4 have convict mothers.
- 17 have cousins convicts.
- 2 have father, cousin, and uncle convicts.

A little over seventeen per cent of our convicts show family crime histories like the above; and where two or more members of one family are convicts, they will nearly always show the distinctive character of the crime class which I have described. They have sprung from a stock conditioned in their mental and physical make-up so badly as to make probable a progeny stamped with imperfections.

Are they fully responsible beings? They are held as amenable to the law as the other class of criminals not by nature such. Responsibility is only questioned when their crimes reach the high degree of murder. The one who has the support of a crime-class history and family record can nearly always be adjudged insane and so escape the penalty of the law.

The study of the histories of members of this part of our community cursed with inherited abnormal mental and moral faculties through several generations—as has been repeatedly done—shows an unbroken line of criminals. They do much to destroy the moral health of others by the contaminating influence of association.

Should not society stamp out this moral ulcer or morbid growth and stop the perpetuation of the species by

separating this class by *life imprisonment* after repeated convictions to State prisons?

During the past five years, in the capacity of physician to the Eastern State Penitentiary, I have had to deal with about 3,500 convicts for periods of one year—the shortest term of sentence—to others including, and beyond these five years. Of these just about 245 were insane upon reception, 40 had insanity develop during their incarceration, and 20 were guilty of simulating insanity. Among the rest were to be found many feeble-minded, epileptic, idiotic, and many in whom intelligence had but slightly developed and showed little promise of improvement.

The usual method by which an insane man develops into a convict is about as follows: He commits a crime, usually against the person or the property of a fellow-man, is arrested, speedily tried and convicted, his guilt being very evident. In the press and rush of work in our Quarter Sessions Courts, no thought is given to his mental condition or responsibility, and he is quickly landed for a term of years in the penitentiary. Here each individual can receive most careful investigation, and thus his insanity is recognized. Others become members of the penitentiary convict population by having been convicted of murder in the first degree, and afterwards having their death sentence commuted by the Pardon Board to a life of imprisonment.

Many different forms of insanity are noticed among the crime class as elsewhere. The most usual form being (1) delusions of attempts to introduce poison into their food, and thus compass their destruction; (2) bodily torture by electricity applied in some indefinable way or by means of the surrounding air—this latter class rarely recovers, and discernible cerebral lesions are often revealed at autopsies; (3) feelings of personal persecution without cause, on account of which impending bodily violence is often likely at any time to be dealt to them, is frequently noticed. One of the most interesting forms is religious exaltation and a belief in personal conversion, and a call to personal work in converting others, often developed

without apparently having been subjected to any previous religious excitement. Of this class it is not unusual to see a man previously of but little mental capacity, and that of low grade, and with almost no education—if any at all—a history of crime from earliest childhood, and an inheritance of crime tendency and disease inclination, both mental and physical, develop suddenly a condition of religious ecstasy and transport, and familiarity with religious and biblical subjects and with a readiness and ability at argument that is nothing short of marvellous. There seems to come vividly to them every word they have heard uttered or read bearing on religious subjects, most of which they certainly could not have comprehended at the time, and its mental imprint must have been of the feeblest sort. Indeed, I doubt if much of it was heeded at its hearing, or the different thoughts individualized or mentally digested more perfectly than, say for comparison, that induced by the various street noises on an old resident of a busy city thoroughfare. Yet those so diseased can apparently recall each peculiar phrase or text and apply it with striking aptitude in conversation and argument. These cases generally progress to general paresis and dementia, or have developed some form of intracranial inflammatory trouble, often associated with or soon followed by some form of tubercular disease.

Another class, the members of which come under the study of specialists in mental diseases, for the purpose of deciding their legal responsibility, is the homicidal monomaniacs. Members of this class will kill without any cause when the impelling seizure attacks them. They sometimes give no reason for their acts other than that they could not resist the impulse. Again they will claim to have divine order to do so. Afterwards, they sometimes do, and at others do not, appreciate the enormity of their crimes.

Insane criminals do not always commit crime as a result of their diseased minds, but their lives have been filled with crime long before insanity developed itself, and if their vicious excesses and irregularities do not break

the physical constitution, often of brute-like endurance, some part must naturally weaken, and mental disease is thus established. They are not criminal because of insanity, but independent of it, and if not insane would still be equally criminal, their crimes during their insane period being the same as repeatedly committed before the slightest symptom of it was apparent.

The simulating of insanity is rarely met with in the experience of the general medical practitioner. In fact, it is so rare as to be seldom met with outside of prisons, the army, or navy, doubtless on account of there being hardly any benefit to be derived from its indulgence elsewhere. I do not believe it would be possible to make the physician or specialist in nervous diseases believe how deceptively an ignorant, illiterate criminal can play the part of a lunatic, unless he has had the opportunity of observing it in practice as I have. I have noted about twenty cases among the convicts in which the effort was of sufficiently deceptive appearance to have puzzled or deceived any physician. These efforts have lasted from days to many months, and in a number of them I could scarcely believe they were not true cases of insanity until they confessed their simulation. I should here explain that the object in being thought insane by convicts is the hope that by that means they may be taken from their separate solitary confinement, and be given work in the large outside yard within the penitentiary walls. If their sentences are long, they hope to be removed to asylums for the insane, which would be a great improvement on hard labor and penitentiary food. Such a removal would also give them much better chances for escape.

I have referred to the wonderful cunning of this crime class. Were their intelligence to be measured by this part of their mental development, they would rank with men of marked ability in the learned professions. One of these men might never be able to learn to make a good shoe, but could easily plan a most intricate piece of burglary work, and subsequently give a plausible answer to every inveigling inquiry without criminating himself.

Those simulating insanity never claim to be insane ; they usually in the first place threaten suicide, and if that, or a safe pretence at its attempt does not give them their desideratum, they put into practice what to them is considered unquestionable evidence of insanity. It occasionally occurs that a man simulating insanity so taxes his mind by a continuous and straining effort that a true pathological condition is induced, often portraying a different form of mental disease from that simulated. Again, in time, the bogus delusions may become truly insane ones, like an oft-told lie, with continued effort to make it appear as a verity, they come to believe in it themselves, regardless of its improbability and preposterousness. Such cases, after regaining mental health, have assured me that the delusion for a time was real with them. The possibility of gaining life when jeopardized, or possibly acquittal when being tried for murder, induces much and often most deceptive simulation. In the history of these cases the most learned specialists in psychology, lacking only experience among the criminal class, are often of directly different views in diagnosis. I am much inclined to think that it is scarcely possible for specialists in mental diseases to gain enough insight into these kinds of cases in the often trivial examination given them. Also that sufficient weight is not given to prison-keepers' opinions, having them in charge for prolonged periods, and so enabled to study them constantly.

The following cases which occurred in our prison should be of value in demonstrating the perfection possible in simulating mental disease by a criminal. The first is the more important on account of the ability and high standing of the specialists who were so completely deceived by him. The facts are as follows :

Convict A., a natural crime-class man, of German birth, about 35 years old, a thief of the horse-stealing variety, was received from one of the counties to serve a sentence of twelve years. During the first month he behaved very well, when one day suddenly he seemed to have an attack of mania, and violent actions, such as are seen in true de-

lusalional lunatics. He became destructive, breaking his cell furniture, etc. This lasted for about an hour, and was followed by a claim that he knew secret harm was being done to him by the officers, and to the belief that his wife and relatives were being subjected to improper treatment. These false beliefs he adhered to with such apparent honesty, in opposition to all denials of those in authority, that he would have deceived almost any one but a veteran in dealing with the criminal class.

His condition grew no better and no less uncertain. A man was placed in charge of him exclusively. He was during all favorable days given the liberty of the large yard, under the care of his guard. He adhered to his delusions with occasional outbursts of violence toward the officials. After attacking them he was always brought under subjection without bodily injury or punishment being done him, being by many thought truly insane. He grew worse and more dangerous, and frequently so violent and destructive that it became necessary after a few months to keep him in a cell devoid of all furniture but his bedding. His habits became filthy in the extreme. Lewdness, to which he had long been addicted, became much more open, and probably more frequent. True dementia seemed to have become established. Looking up at one spot for hours with, at times, insane mutterings, was one of his actions, also smiting his fists and head against the stone walls with great violence for many minutes at a time. He bumped his nates against the iron cross-barred door till the skin was denuded and flesh much lacerated. In appearance he became a most pitiable object.

Eight months after the start of this masquerade, his wife, by legal assistance, had a committee in lunacy appointed by the court to have him adjudged insane and removed to an asylum.

The law had not yet been changed, so the committee consisted of the District Attorney of Philadelphia and two physicians.

The medical men were the principal physicians of two



of our largest asylums for the insane, and were highly learned and notably careful observers in their specialty. The committee visited the man in his cell, and made a prolonged study of him, as they knew his insanity was doubted by some of the officers. They familiarized themselves with all the evidence in detail. The examination convinced them of his undoubted insanity. They asked the opinion of each officer examined as to his insanity.

From those opposing their views they required their expression of opinions under oath. So fully were they assured of his insanity that they thought an opposing opinion must be due to some foreign influence or special interest in the case. As demonstrating how firm they were in the belief as to his complete mental incapacity, they told one of the chiefs of the departments in the penitentiary that although he seemed to be an intelligent man, yet they questioned his knowledge as to what an insane man really was, if he would swear that he doubted this man's insanity. As the committee signed the certificate declaring him insane, it was expected that he would be removed by order of the court in about one week, the usual time. The man got no better, and at times violently attacked the keepers. A month passed, but by some neglect or oversight of the court the order for removal had not arrived. One day, two keepers went in to clean his cell and change his clothing. He struck one of them, and attacked him with great violence. Prior to this he had always been very kindly treated, and had not been the recipient of any punishment; but this time the keeper attacked was so angered that he retaliated, and subjected him to a sound drubbing.

This keeper was one who testified to a belief in his feigning insanity. One hour after the chastisement, the convict knocked on his door, and called for this keeper to come to him. As soon as he answered his summons, the malingerer addressed him in these words:

"Mr. Mc——, I am sick and tired of playing crazy. If you will put me in a cell with things in it, I will always

behave myself as well as any man in the house, and always do my work."

This request was at once complied with, and he kept his word, and during the remaining six years of his imprisonment he was one of the best behaved men in the institution, and always perfectly sane. The judge being notified, no order was made for his removal, and he was pardoned after serving seven years.

I do not know if the physicians who examined him ever heard of the result after signing the certificate of his insanity.

If present this evening, they may learn for the first time how completely they were deceived by an ignorant German, naturally criminal. The last time I heard of the man he was driving a cart in a brick yard in the city.

He made the following statement as to why he feigned insanity: "There seemed no end to a twelve years' sentence, if I looked forward to it. It was terrible; and fearing I could not live it out, I thought if I played crazy I would be sent to an asylum, and would get quickly cured and discharged, or could escape easily; but the doctors would not send me." Every statement in regard to this case can be supported by the affidavits of different officials in the penitentiary.

The gross miscarriage of justice and wrong done to society in the Emma Bickel case need only to be mentioned. Such cases cause the finger of shame to be pointed at our profession.

Several of the murderers executed in our city recently have been declared insane by experts. In nearly every case delusion was the form of the aberration. Careful observation has taught that if a man is truly insane, his insanity and delusions will continue up to the hour of his execution. If it is feigned, and all hope gone, every effort of saving his life a failure, and execution inevitable, on the last two days of his life his delusion and insanity disappear. Autopsies often reveal cerebral lesion in the first cases, but only characteristic criminal brains in the last-mentioned.

CASE B.—An Englishman was received as a convict in the penitentiary a few months ago, and indications of insanity were soon evident. Different medical friends whom I requested to see him decided him to be insane, but I did not believe it. He got worse, and eventually I was one day summoned to see him, "as he had a stroke of apoplexy, and one side was entirely paralyzed."

They thought the man about to die. His simulation of the paralysis was perfect, excepting that the wrong eye was affected.

I was much in doubt, but decided to try a test on him. I explained in his hearing to those present that it was acute congestion of the brain, and that I would, as the only hope, have to chloroform him, and with a hot iron burn the bone at the base of the brain.

I hastily got the cautery ready and red-hot, and proceeded to etherize him. The heaviest coma was apparent, puffing and all; pin jags produced no response. Suddenly, when he had inhaled enough ether to be pretty well intoxicated, and to feel himself losing consciousness, the paralyzed arm was quickly raised, and the ether was dragged away by it. He then shouted: "Take away the damned stuff! I believe you *would* burn me."

He sprang from the bed to defend himself, and that was the end of both paralysis and insanity. He has since been a good prisoner.

An educated, intelligent farmer from one of our northern counties was received two weeks ago to undergo a sentence of eight years for murder. His mental soundness being questioned, he was not hung. He seemed in a very bad condition mentally, and had apparently a distressing pain in his heart. The sheriff had a physician accompany him on his journey to the penitentiary. He seemed in an agony of pain, had eaten nothing for days, and took nothing for the first two days he was with us. He stared blankly, became filthy, lay on the floor day and night, and would only speak in unintelligible mutterings. He would roll off on the floor as often as he was put in bed. Pulse, tongue, and temperature were normal.

He seemed almost devoid of mental power. I thought he was malingering, and decided to break it up at once. I had him picked up, and thrown pretty violently on the bed, and given a rather good shaking up.

I told him, in a way calculated to make him think I meant it, that I would have no more of his nonsense, that I knew him to be perfectly sane and well, and that if he did not get up and behave himself, I would have him roughly handled. He was then left till the next day, when he was up and about, but full of insane delusions and talk. He wanted beef tea, chicken broth, and oysters. He only received another application of the tongue treatment, and was left to think over it. His delusions lasted all day. When talking to the officers, he did not seem to know where he was, had his farm and friends in his cell, etc., etc. That evening I visited him, and assured him he could gain nothing by feigning insanity, as we always kept our insane convicts during their sentence, so removal on that score was out of the question; that if he was willing to start afresh and behave himself, all would be well, and he would fare much better. He thought over it a little while, and then consented, and has not since shown the slightest indication of insanity or physical pain or disease. He proves to be quite an intelligent man, and is much ashamed of his behavior since it failed in its purpose.

Dr. J. William White, my predecessor as physician to the penitentiary, in one of our annual reports cites a case in which a man carried on his malingering to an excessive degree. He eventually became apparently entirely demented, and addicted to the most filthy and loathsome habits of uncleanness. He afterwards acknowledged it was play, and his insanity ceased.

Many more cases might be detailed, but I will only add a few remarks about a case with which you are all more or less familiar, and which has been frequently talked of on this floor. I refer to the Taylor case. There is much to be gained by a dispassionate study of this case, and it is very seriously to be regretted that its argument

has produced bad feelings at times, which invariably blinds conviction.

The following facts are interesting. Not one person, either a member of our profession or of the laity, including his officers in this and the Moyamensing prison, after prolonged opportunity to study his case mentally, day after day, believed that he was *really insane*. The exception to this may be the moral instructor to the penitentiary, who saw him once a month at his cell door. Like all other cases mentioned, he had no delusions or insanity about him during the last two or three days of his life, when all hope of a respite or commutation to a life sentence had disappeared. At one of our meetings here, we were led to expect grave cerebral lesion as probable in his case. The blackboard was used to demonstrate this theory. The autopsy revealed no such lesion, but only a brain of low order and characteristically criminal. His crime had been only what was attempted by him repeatedly, through prior years, before any dream of delusions or insanity had been associated with his career. He was always a crime-class man, doing violence on the slightest provocation. His moral nature had reverted to the savage age of man, proven by his life acts and a study of his intracranial contents. He was a typical son of his kind, an offspring of the noted "Reading hose gang" of homicidal outlaws, and he and his associates were of one clique.

They were only the younger representatives of these notorious outlaws. A few days before his execution, when his last life chance was gone, and he was apprised of it, he told me, in the presence of his keeper, that he knew he was rightly convicted, and well deserved hanging. He said: "I killed Doran for nothing but cussedness, and only wish I had been hung before Doran was killed!" He said all his life had been wrong, and he would be better out of the world. He said also he was sorry he had ever charged Doran and me with what he knew was not true.

When a prisoner's insanity is really questionable, and either the prosecution or the defense, instead of the court,

can have experts to bear testimony pro and con as to his mental condition, so long will they be looked on by jurymen as partisans in the case, and the influence of their opinion reduced to a minimum. It is most to be regretted that experts, in support of their belief, let themselves become such bitter partisans, and impose *their opinion* as *facts positive* on every one and in every place possible.

Expert medical testimony should be the most influential possible, and the responsibility for its not being so lies with the experts themselves. They should use such care as to prevent it so degenerating in the estimation of the laity that the finger of ridicule can so often be pointed at it with some success.

## CASES ILLUSTRATING VARIOUS FORMS OF HEMIANOPSIA AND OTHER IRREGULARITIES IN THE FIELD OF VISION.

By G. E. DE SCHWEINITZ, M.D.

**I**N the JOURNAL OF NERVOUS AND MENTAL DISEASE for January, 1886, Dr. E. C. Seguin contributed an important paper entitled, "A Contribution to the Pathology of Hemianopsia of Central Origin (Cortex Hemianopsia);" important because the conclusions there formulated were based only upon post-mortem determination. In the same Journal for August, 1886, there is another paper from the pen of Dr. Seguin, entitled, "A Clinical Study of Lateral Hemianopsia," in which nine cases of this affection without post-mortem determinations are recorded. In it Dr. Seguin remarks, "The state of our knowledge is such as to render every new case of lateral hemianopsia with autopsy of extreme interest and scientific value; yet it does not, I think, render quite useless the publication of cases without post-mortem study. A number of points in the clinical history and symptom-grouping of hemianopsia may be illustrated by such cases and the diagnosis *intra vitam* of other cases facilitated." Somewhat in the same spirit I bring before you to-night these cases which illustrate various forms of hemianopsia as well as other irregularities in the fields of vision.

CASE I.—Daniel G., æt. 40. Patient of Dr. H. C. Wood, in the Nervous Wards of the University Hospital. Until eight years ago health was good; he was then attacked with pains in the legs which were said to be rheumatic, and which gradually grew worse until the power of his lower limbs was lost. He has had three attacks of

morbid sleep, the last one of which began in May, 1885, and continued until the following September. During this time he lay motionless, with his eyes closed and occasionally for days at a time apparently entirely unconscious. Usually he could be aroused, but would quickly return to his somnolent state. In September he awakened, and then violent frontal and occipital headaches and spells of vomiting were the marked features of the case. Under enormous doses of iodide of potash and mercury the general condition improved, but when he left the hospital in November his headaches remained.

*Examination.*—A large-framed man, with pallid un-

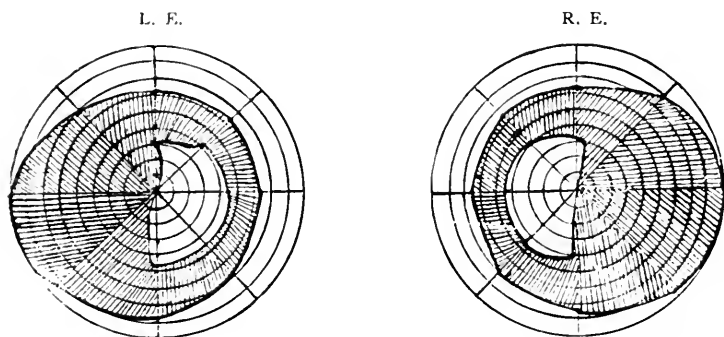


FIG. 1, CASE I.—Bitemporal hemianopsia. The outer boundary of shading represents the limits of the normal field; the shading where vision was lost.

healthy skin. Intellectual faculties good. No paralyses, but the gait labored, like that of a tired man. Vision in each eye  $\frac{1}{2}$ ; both optic nerves gray, with shallow atrophic excavations. There was typical *bilateral temporal hemianopsia* with concentric limitation of the preserved fields. The dividing line almost touched the fixing point, but above this inclined to the right, while below it inclined to the left.

*Diagnosis.*—As far as our present knowledge goes, this form of hemianopsia can only be produced by a lesion of the optic chiasm in its anterior or posterior angles. The patient denied all venereal history, but in spite of this the lesion was probably a syphilitic deposit, otherwise he



would have been unable to take the enormous doses of antisyphilitic remedies which were given, taken, moreover, as they were, with distinct benefit to himself.

CASE II.———, aged about 55, colored. A patient under the care of Dr. H. C. Wood, in the Nervous Wards of the Philadelphia Hospital. I am unacquainted with his previous history, except that he had syphilis.

*Examination.*—He had epileptic attacks, Jacksonian in type, partial loss of hearing on side, demonstrable loss of taste and smell, partial hemianæsthesia and hemiplegia. Pupils were of medium size and sluggish in action. Vision, in right eye, counts fingers; in the left eye, only light percep-

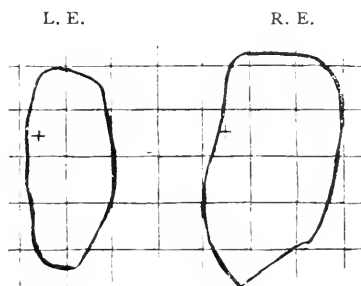


FIG. 2, CASE II.—Bilateral temporal hemianopsia, shape of fields simply represented, the left was taken with a candle. Drawn from memory. Tracing under R. E. inverted by mistake.

tion. Both optic nerves atrophic and devoid of capillarity. There was complete *bilateral temporal hemianopsia*.

The patient died, and at the post-mortem examination there was found a band-like gumma which stretched across the anterior end of the pons and reached to the cribriform space. One end of the tumor was thicker and heavier than the other. The corpora quadrigemina and optic tract, as well as the auditory nerve, were involved on one side. ("Nervous Diseases and their Diagnosis," H. C. Wood; p. 271.)

CASE III.—Ellen C., æt. 45. Presented herself last August in the Dispensary for Diseases of the Eye in the University Hospital to obtain an order for presbyopic glasses. Seven months prior to this date she had a partial

left-sided paralysis. Otherwise she had had no noticeable recent illness.

*Examination.*—The hemiplegia had passed away, although there was impaired muscular power upon that side of the body. The grip was weakened, as tested with the hand, and in walking it was noticed that she “favored” the left leg. There was no demonstrable anæsthesia. The left breast was invaded by a large, painless growth (cystosarcoma?). No cardiac murmurs were detected, but the aortic valves closed with a metallic snap. Vision in each eye  $\frac{20}{xxv}$  and with suitable presbyopic glasses she read easily. With the exception of broadthening of the scleral

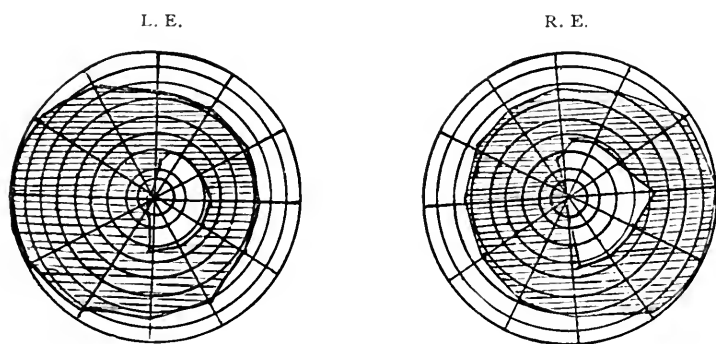


FIG. 3, CASE III.—Left lateral hemianopsia. The outer boundary of the shading represents the limits of the normal fields and the shading the areas where vision was lost.

rings, there were no changes in the optic nerves. The central circulation was normal, the choroid vessels were exposed on the periphery. There was *left lateral hemianopsia* and constriction of the preserved fields, this constriction being the most marked in the left eye. The dividing line passed almost through the fixing point, but sloped slightly above and below. She was practically unconscious of the visual defect, although on questioning she mentioned “that sometimes when sewing the thread looked as if it had been cut in half.”

*Diagnosis.*—A lesion situated in the inferior parietal lobule and angular gyrus may perhaps explain this case, and it would then belong to the class of cases explained

by No. 6 of Seguin's rules. It is interesting to note the fact that the patient had a morbid growth of the breast, although I am inclined to think a hemorrhage in the region named or else in the occipital lobe was the lesion.

CASE IV.—Henry J. McG., æt. 49. A patient under the care of Dr. H. C. Wood in the University Hospital. Has had syphilis and been a hard drinker. In 1878, after a sleepy feeling, awoke, and found he had lost his memory for words, and forgotten French and German, with which languages he had previously been conversant. He had partially lost control of his *right* side. Treatment with iodide of potash produced marked improvement, and he

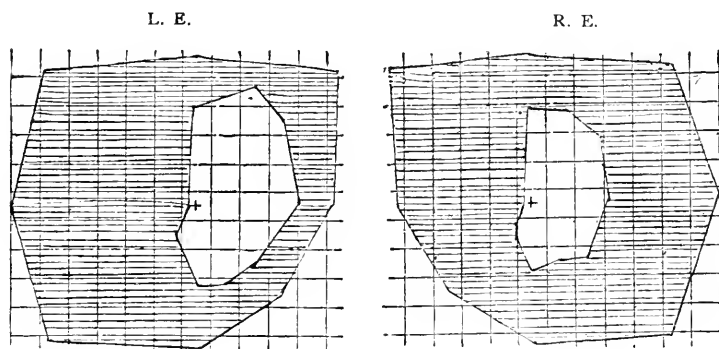


FIG. 4, CASE IV.—Left lateral hemianopsia. Fields taken on a ruled blackboard. The outer limits of the shading represent the boundaries of the fields assumed to be normal, the shading the areas of lost vision.

remained under treatment for two years. April 25th, 1886, while at dinner, a feeling like sea-sickness came over him, followed by maniacal excitement and loss of power on *left* side of his body, so that he was unable to walk or stand alone.

*Examination.*—April 29th, 1886. The memory for words was poor, but chose such words which in sound resembled those he desired to enunciate; thus he said "struck" for "stroke," "intelligently" for "intentionally." Face was drawn to the right side, the limbs were rigid and movements exaggerated. The patellar reflexes were increased, especially on the left side. There was no ankle

clonus. Felt the prick of a pin at any point in the arms and legs, but could not distinguish two points in the longitudinal axes of arms or legs, even when many inches apart, five on the forearm and nine on the leg. June 1st, 1886, examination of eyes. Vision in R. E.  $\frac{20}{xx}$  ( $\frac{2}{3}$ ); in left eye  $\frac{20}{l}$  ( $\frac{1}{2}$ ). Nerves gray and horizontally oval. Retina hazy. *Left lateral hemianopsia*, with some contraction of the remaining fields. The dividing line was slightly in advance, *i. e.*, to the left of fixation, and just below the horizontal plane in each eye bent into an abrupt angle. The pupils were normal in their reactions to light and shade and to convergence, and were of equal size. November 30th, 1886. Has been able to walk alone; the gait was spastic and somewhat ataxic. February 10th, 1887. Eye examination was repeated and fields and appearances of the optic discs found to be the same as they were at the original examination.

*Diagnosis.*—The presence of lateral hemianopsia, hemiplegia (spastic after a time), and aphasia would naturally associate the lesion in this case with the motor zone and the convolutions at the end of the Sylvian fissure as in case by Westphal (Case 26, Seguin's list). But the clinical history shows that there were two attacks of hemiplegia, eight years apart, in the first of which there was right-sided palsy and aphasia, and in the second of which there was left-sided paralysis. It was only after the second attack that the eyes were examined, and the hemianopsia then found to be left lateral, and consequently it must have been the result of the second attack.

CASE V.—W. S., æt. 29. A patient under the care of Dr. H. C. Wood in the Nervous Wards of the University Hospital. He was well until his twenty-first year, when he began to have asthma. About ten months before admission began to be morbidly sleepy, and one month after this had a convulsion, during which, according to the statement of his wife, he "worked all over." He had a number of similar attacks until September, 1886, when, following such a seizure, he became irrational and violent, remaining so for twenty-four hours. During

the preceding April had an attack, when the convulsive movements began on the right side (leg and arm), and after this the right side was paralyzed for a number of hours. Denied syphilis. Patellar reflexes absent.

*Examination.*—Right pupil slightly larger than left. Both react to convergence, but not to light. There was constantly present an irregular tremor or chorea of the right arm and sometimes of the right leg, *rarely* in the left leg. The speech was slow and hesitating, the memory impaired. His vision was, O. D.  $\frac{20}{XXV}$ , O. S.  $\frac{20}{XX}$ ; optic nerves gray-red; edges obscured; veins full and tortuous. The *left fields* were dark, the right fields much

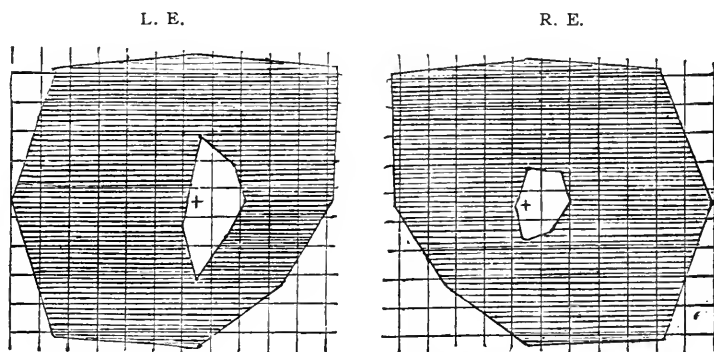


FIG. 5, CASE V.—Left lateral hemianopsia. The tracings and shading as in the other diagrams. Marked contraction of the visual field.

contracted, most markedly on the right side. The dividing line was irregular, passing distinctly in advance of the fixing point. After a course of mercurials, he improved somewhat, but reported occasional fits, usually right-sided, but occasionally left-sided. This improvement continued until November, 1886, when he grew worse. The seizures appeared with increased frequency, he became noisy and restless at night; the muscles of the face constantly twitched. He was often observed to masturbate. The urine had a specific gravity of 1.020, was slightly albuminous, but there was no sugar present. The sediment contained oxalate of lime crystals and spermatozooids. At times he exhibited typical delirium of gran-

deur. Finally he became so violent that it was necessary to send him to an asylum.

*Diagnosis.*—It seems quite evident from the history that no single lesion in this case produced the symptoms, and, moreover, that there was an affection of the cord as well as of the brain. It is most probable that a deposit, probably syphilitic meningitis, pressed upon the right optic tract, paralyzing the right temporal and the left nasal retina. This becomes the more assured when it is remembered that there were optic neuritis and pupillary immobility.

CASE VI.—John G—, æt 40. A patient of Dr. Osler, referred to me for examination. During the war sustained

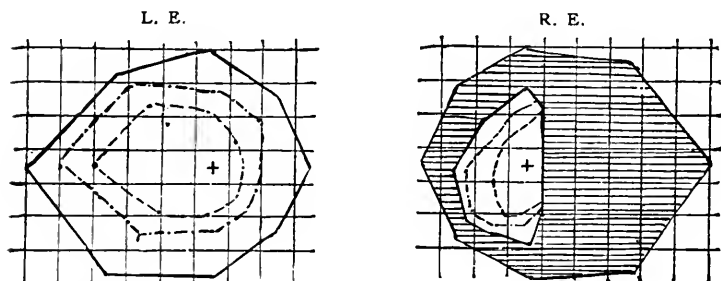


FIG. 6, CASE VI.—Unilateral temporal hemianopsia. Fields of vision for white, blue, and red. The shading in the left field represents where vision was wanting.

a fracture of the skull in the region of the left parietal bone.

*Examination.*—In the region of this bone, a distinct depression was observed. He was somewhat dull of comprehension, and subject to attacks of epilepsy. Pupils equal and normal. Vision O. D.  $\frac{20}{20}$ , O. S.  $\frac{20}{20}$ . The field of vision for form and colors in the right eye about normal. In the left eye, the larger part of the temporal field was dark. The dividing line passed to the left of fixation in a straight line. There were no abnormalities in the eye-grounds. The scleral rings on both sides were broadened.

*Diagnosis.*—It seems not improbable that this case was the result of the fracture which may have injured the bones of the orbit and the fibres of the left optic nerve

which pass to the nasal half of the retina. Dr. C. S. Bull (Trans. Ophthalmological Soc., 1885) has reported a very similar case.

CASE VII.—Mrs. L. W., æt. 37. Patient referred to Dr. Randall for examination in November 1886, and two months later examined by myself at the University Hospital. Recent general health has been poor, frequent right-sided neuralgia and “stomach trouble,” both of which, however, improved under treatment. Has had five children (two husbands), all healthy, youngest twenty months old. Eighteen months after birth of fourth child, four years ago, was seized with temporary right-sided hemiple-

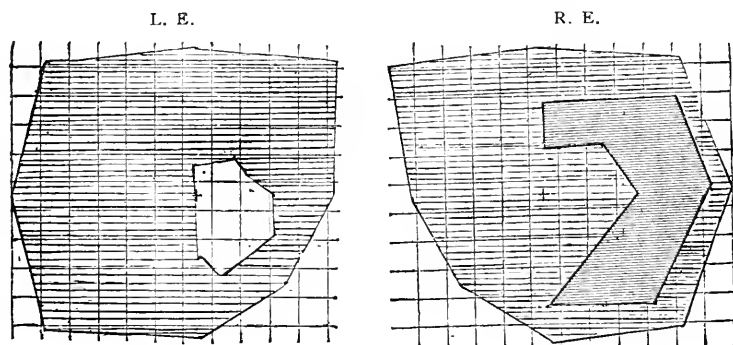


FIG. 7, CASE VII.—Temporal hemianopsia in L. E. In R. E. complete blindness, except an irregular patch of faint light perception in the right field, represented in diagram by the close shading; the light shading represents where vision was lost.

gia, which passed away under use of electricity. Twice since then has had attacks of unconsciousness, followed by convulsions.

*Examination.*—Small woman, of rather ruddy complexion, which in the cold turned blue. Cardiac palpitation was easily induced, and there was a soft basal murmur. The muscular power of right side was impaired, but there was no palsy. The tongue was tremulous and turned to the left side. The patellar reflexes were prompt, more so on the left side. The left hand was colder to the touch than the right, and pain from prick of pin was said to be the more readily appreciated on the right side, but the opposite side was not anæsthetic. Pupils unequal, larger on the right

side. Vision in O. D. absent, possibly faint quantitative perception of light; in the left  $\frac{20}{xxx}$  ( $\frac{2}{3}$ ). Optic discs oval, shelving excavations, full and tortuous retinal vessels. The appearances on both sides almost identical. In the right eye no field even for light, except a patch on the temporal side where the light was doubtfully perceived. In the left there was total, left (temporal) hemianopsia, the dividing line passed directly through the fixing point and did not incline either above or below it.

*Diagnosis.*—Dr. Gowers ("Med. Ophthalmoscopy," 2d Ed., Case 30, p. 311) records the case of a boy who, one year after a fracture of the skull, suddenly had a fit, after which the left side became paralyzed, the left eye blind, and there was loss of the left half of the field of vision in the right eye. The case did not terminate fatally, but in commenting upon it, Dr. Gowers says: "The only theory on which the affection of sight can be explained is that of Charcot, which supposes the semi-decussation in the chiasma to be supplemented by another farther back, so that each eye becomes represented in the opposite hemisphere, although only half of each eye is represented in each optic tract. Charcot's theory is still an hypothesis only, but it is noteworthy that on this theory a lesion about the right corpora geniculata, so extensive as to destroy the fibres which come from the right optic tract (and the right halves of each retina=left halves of the fields), and also to destroy the decussating fibres supposed to come by the corpora quadrigemina from the left optic tract, would give precisely the condition present in this case, the only optic fibres undestroyed being those from the inner half of the right retina (outer half of the field) which pass by the left optic tract to the left hemisphere . . . A lesion so placed as to explain these (the cerebral symptoms) would probably involve or be beneath the lower part of the parietal lobe and angular gyrus." Charcot's theory and diagram are now quite universally rejected, I believe; but even were they proven to be correct, and were we to reason for this case as Dr. Gowers did for his, substituting for the word *left* the word *right*,



the explanation would not suffice. In his case there was left hemiplegia, left amblyopia, and loss of vision in the left half of the field of the right eye; hence, Charcot's theory being accepted, his reasoning as to the seat of the lesion is most accurate. In this case, however, there was right hemiplegia (partial), right amblyopia, but loss of vision in *left* half of the field of vision of the right eye, and not in right half of the field, as it would have to be were we to similarly place the lesion. On the other hand, a lesion so situated at the chiasma that it had destroyed all the fibres of the right optic nerve and damaged the inner half of the left, thus paralyzing the nasal half of left retina, making dark the temporal half of the field, would exactly explain the affection of sight. Whether a lesion so situated would also explain the cerebral symptoms I am in doubt, as I am also in doubt as to what the nature of such a lesion would be. Hysteria, as a possible causative factor, has been thought of, but this case does not correspond in its features to one of hysterical amblyopia.

CASE VIII.—John F., æt. about 30, a patient under the care of Dr. H. C. Wood, both in the Philadelphia and University Hospitals. I am unfortunately unable to give any detailed history, as the notes have been mislaid.

*Examination.*—Complete right-sided hemiplegia, hemianæsthesia, and aphasia. R. E. vision 0. In L. E.  $\frac{20}{xxx}$ . Right optic nerve gray-green, arteries small, veins full and tortuous, scleral ring breadthened all around. In L. E., small nerve, with breadthened scleral ring, edges of disc slightly hazy. Veins full and tortuous. There was apparently lateral hemianopsia; the field in the right eye was taken with a candle. It was then thought that either there was a double lesion or that a large clot in the lenticular nucleus might by pressure paralyze on one side Broca's convolution, and on the other the knee of the internal capsule. I repeated the examination in this case this winter, about eight months after the original examination, and then found vision in R. E. 0, in L. E.  $\frac{20}{L}$ . The fields were as in the diagrams, viz., in the R. E. absolutely dark, and in the L. E. only a small irregular

patch of preserved vision on the temporal side. I am inclined hence to think that there was no hemianopsia in this case, but that there were markedly irregular fields, as in neuritis; that the condition of the optic nerves was one of consecutive atrophy, the neuritis having entirely subsided in the R. E., and the atrophy become complete, while in the L. E. the process was not so far advanced, but that it will go on and the man will eventually become blind. This case was demonstrated by Dr. Wood before the Philadelphia County Medical Society, and has also furnished the material for one of his clinical demonstrations before the medical class of the University.

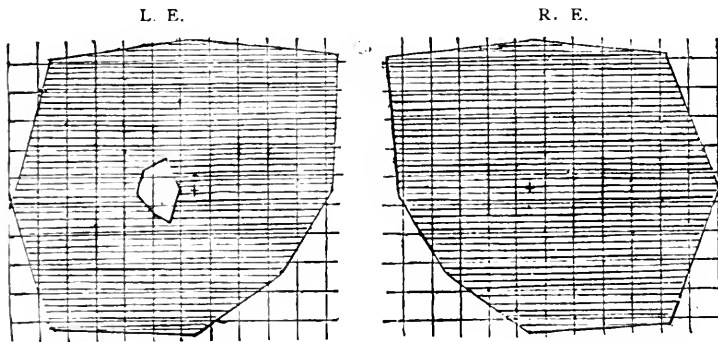


FIG. 8, Case VIII.—R. E. complete loss of vision, field entirely dark. L. E. small area of preserved field on the temporal side. Shading represents areas of lost vision, and the limits of the shading the boundaries of the fields assumed to be normal.

I will call attention to the following points:

1. The optic nerves were atrophic in the two cases of bitemporal hemianopsia, and there was slight but well-marked optic neuritis in one of the cases of left lateral hemianopsia (Case V.). In these cases the lesion was quite certainly at the base of the brain. There were no changes in the fundus in one of the cases of lateral hemianopsia, in the remaining one the optic nerves were gray, but there was fairly good central vision. In the case of so-called unilateral temporal hemianopsia, there were no fundus changes, and central vision was normal.

2. In no case was the hemiopic pupillary reaction observed.

3. In all the cases the dividing line passed with some irregularity, *i. e.*, slightly in advance of the fixing point, with the single exception of Case VII., where it passed directly through the fixing point.

4. One patient (Case III.) was practically unconscious of the visual defect.

## A RESUMÉ OF RECENT TECHNICAL METHODS FOR THE NERVOUS SYSTEM.

BY IRA VAN GIESON, M.D.,

ASSISTANT AT THE LABORATORY OF THE ALUMNI ASSOCIATION OF THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK.

### GOLGI'S METHODS.<sup>1</sup>—I. *Silver Process.*

Small pieces (one to two cubic centimetres) are placed in a two-per-cent solution of potassium bichromate (or Müller's fluid) containing camphor. The concentration is increased at each renewal of the fluid up to five per cent. The length of time of the hardening varies according to the amount of the material, the concentration of the solution, and the temperature. A constant temperature (20–25° Cent.) is advantageous. If the thermostat is not used, in summer, portions of the brain are hardened fifteen to twenty, rarely forty to fifty days, and in winter five to six, to sixteen weeks. To avoid all post-mortem changes, the author injects a two-per-cent solution of bichromate, containing five to six per cent of gelatin, into the carotids. The hardening process may be hastened by replacing a part of the hardening fluid by Erlicki's fluid in increasing proportions. A still more rapid procedure consists in the after-hardening in a mixture of eight parts of a two-and-one-half-per-cent solution of bichromate and two parts of a one-per-cent solution of osmic acid.

The hardened pieces are placed in a large volume of a three-fourths of one-per-cent solution of nitrate of silver,

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<sup>1</sup> C. Golgi, "Recherches sur l'histologie des centres nerveux." Archives italiennes de Biologie, Vols. iii., iv., 1883.

C. Golgi, "Sulla fina anatomia degli organi centrali del sistema nervoso." Review in Neurolog. Centralblatt, No. 13, 1886.

which must be renewed at intervals until the chromate of silver precipitate no longer forms. If the hardening does not seem complete, a one-half-per-cent silver solution should be used. If the hardening has been protracted, the silver solution is increased to a strength of one per cent. The blocks remain in the silver solution twenty-four to thirty hours; a longer immersion is not usually injurious. After cutting, the sections are carefully washed in alcohol, cleared first in creasote and then in turpentine. If the sections are carefully washed, they need not be protected from light. During the hardening, it is advisable to test portions occasionally with the silver solution, for the method is somewhat uncertain.

2. *The Sublimate Process.*—The same results may be obtained if the hardened pieces are immersed in a one-half-per-cent-solution of bichloride of mercury instead of the silver solution. The immersion in the sublimate solution requires several weeks. This method has the advantage that very large masses, even the entire brain, may be stained *in toto*, thus facilitating the preparation of serial sections.

The results accomplished by Golgi's methods have recently been highly commended by Forel.<sup>1</sup> The astonishing selective affinity that these metallic combinations exhibit for the ganglion-cell with all of its processes, and for the intricate network derived from the axis-cylinder process of certain ganglion-cells, and for the transitional area from the ganglion-cell to the nerve-fibre opens new histological and pathological territories. Perhaps some of the incomplete chapters in cortical pathology will receive contributions from this method, and a re-examination by this process is required of cases in which delicate changes in the ganglion-cells are suspected, but which have eluded the previous methods.

A great advantage of Golgi's process is the contrast afforded between the colored and the uncolored elements, as the ganglion-cells and their immediate adnexa are sharply

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<sup>1</sup> Prof. A. Forel, "Einige hirnanatomische Betrachtungen und Ergebnisse." Archiv f. Psychiatrie, Band xviii., No. 1.

delineated in black against a faint yellowish back-ground. This black color in the silver process is probably due to a precipitate of chromate of silver; in the sublimate method, to one of the oxides of mercury. This contrast of color renders thick sections rather more useful than thin ones, for in the latter many of the processes of an individual cell may be lost. A peculiar feature of this method is its property of staining only here and there scattered and isolated elements in the section—a less confusing picture than if the whole section were uniformly stained.

Bleuler<sup>1</sup> has succeeded with the stain and has obtained fac-similes of many of Golgi's plates. A resumé of Golgi's remarkable propositions concerning the ganglion-cells, many of which are sustained by Forel, may be found in Forel's<sup>2</sup> paper. Pal,<sup>3</sup> in discussing the results obtained by a modification of Golgi's methods, observes that the neuroglia-cells stain in the same way as the ganglion-cells, and that the neuroglia-cells of the white substance react the best. Pal obtained the best results in the cortex; the cerebellar sections were less successful, and the results in the cord were almost entirely unsuccessful. The one disadvantage of the method is its uncertain results.

*Pal's Modification of Golgi's Methods.*—1. If the sections prepared by the sublimate process are inspected with the naked eye, they contain whitish, opaque spots. The sections are immersed in a one-half to one-fourth-per-cent solution of sodium sulphide until the spots become blackened. The sections are then washed and may subsequently be advantageously stained with magdala-red.

2. The silver preparations may be treated in the same manner. The sections are placed in the sulphide of sodium solution, which gives a black sulphide of silver precipitate in the ganglion-cells. The author claims that this precipitate is more stable than that of chromate of silver occurring in Golgi's original process. These prepara-

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<sup>1</sup> Correspondenzbl. f. Schweizer Aerzte, March 15th, 1886.

<sup>2</sup> L. c.

<sup>3</sup> J. Pal, "Ein Beitrag zur Nervenfärbentechnik;" *Medicin. Jahrbücher der K. K. Gesellschaft*, 1886, No. 9.

tions do not show the network of the processes as well as the modified sublimate method.

**PAL'S MODIFICATION OF WEIGERT'S METHOD.**—Pal proposes a modification of Weigert's method.<sup>1</sup> The copper immersion is dispensed with, and two to three cubic centimetres of lithium carbonate are added to Weigert's hæmatoxylin solution. Sections are stained twenty-four to forty-eight hours. The sections are washed in water; if the sections are not stained a deep blue color, one to two cubic centimetres of lithium carbonate are added to the water used for washing. The sections are then transferred to a one-fourth-per-cent solution of permanganate of potassium for twenty to thirty seconds, where they present the same appearances as in decolorizing with the ordinary method. The specimens are then transferred to the following solution:

Oxalic Acid.....	1
Potassium Sulphite.....	1
Aq. Dest.....	200

for a few seconds. The sections are better adapted for subsequent contrast staining than those obtained by the usual method. If flocculi persist on the surface of the sections while in the oxalic acid mixture, the sections are again rapidly passed through the permanganate solution, and then replaced in the oxalic acid mixture.

Sections prepared by Golgi's methods may be stained by this modified Weigert's method, by placing them for twenty-four hours in a five-per-cent solution of chromic acid previous to staining them in the hæmatoxylin solution. Pal also recommends a one in four hundred aqueous solution of potassium permanganate, for removing the color from all structures except the medullated nerve fibres, in

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<sup>1</sup> Freud (Vienna), reviewing this modification for the *Neurolog. Centralblatt*, March, 1887, recommends it, and observes that the differentiation is sharper than that obtained by the ordinary method. Serial sections between two adherent films of celloidin should be first placed for a few minutes in the prussiate-borax solution of Weigert, and then be subjected to the differentiating solutions of Pal.

sections of the nervous system hardened in one-per-cent osmic acid.

**ALKANNA FOR MYELIN STAINING.**—Achard<sup>1</sup> recommends a concentrated alcoholic solution of the cortical portions of alkanna root for the central nervous system and also for the peripheral nerves. Fragments of the roots are macerated in a relatively large volume of ninety-per-cent alcohol for several days in a well-corked flask, until the solution has a garnet-red tint. The solution must frequently be prepared anew. Specimens hardened by the chrome salts are left in this solution about two hours, avoiding the access of air by using carefully closed dishes. If the hardening has been protracted, the sections should not remain too long in the staining reagent, for the color may become diffuse. The stained sections are rapidly washed in water and mounted in glycerin. The myelin has a deep brown color, and the result is similar to that given by Weigert's method, with the disadvantage, however, that the sections cannot be mounted in balsam, for this procedure removes the color completely. The sections may be stained with carmine first and subsequently with the alkanna.

**CHANGES PRODUCED IN GANGLION CELLS BY HARDENING REAGENTS.**—Trzebinski<sup>2</sup> has studied the changes produced in the ganglion cells of the spinal cord in dogs and rabbits, by using the following reagents:

1. Müller's fluid (five to six weeks), followed by alcohol.
2. Alcohol ninety-six-per-cent.
3. Chromic acid, followed by Müller's fluid or alcohol.
4. Hardening in a ten-per-cent solution of bichloride of mercury for eight days and subsequent hardening in alcohol containing five-per-cent of iodine. Reagents 1 and 3 produce very serious alterations in the contour and finer

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<sup>1</sup> Ch. Achard, "Sur l'emploi de la Teinture d'orcanette dans la technique histologique;" *Archives de Physiologie*, 1887, No. 2.

<sup>2</sup> Dr. S. Trzebinski, "Einiges über die Einwirkung der Härtungsmethoden auf die Beschaffenheit der Ganglienzellen im Rückenmark der Kaninchen und Hunde." *Virchow's Archiv*, Band 107, No. 1.



structure of the cell, which have undoubtedly been ascribed by some authors to pathological processes.

In specimens hardened by reagent 4, these changes are reduced to a minimum. Vacuoles and pale or faintly stained ganglion cells<sup>1</sup> are absent. The pericellular spaces are small and the finer structural details resemble closely the appearances seen in the cells when examined in the fresh condition. The condition of the cells after alcoholic hardening closely resembles the result obtained by the sublimate hardening, and the author indicates a preference for the latter method.

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<sup>1</sup> Dr. F. Kreyssig, Virchow's Archiv, Band 102; H. Koneff, Fortschritt. d. Med., 1886, No. 23.

## Clinical Cases.

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### REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILA- DELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF W. H. WALLACE, M.D., PHYSICIAN  
IN-CHIEF, AND CHARLES K. MILLS, M.D., CONSULTING PHYSICIAN.

#### CASE XIII.—*Paranoia.*

Reported by Dr. Allen J. Smith, Assistant Physician.

P. J. O., white, æt. 48, laborer, a native of Ireland, was brought to the hospital March 23d, 1887, having been transferred by order of the Lunacy Committee of the Board of Charities from the State Asylum at Norristown, Pa. There was no known insanity in his family, near or remote, nor any history of hereditary diseases. Personally he had never been subject to serious illnesses, with a single exception. He denied all venereal troubles, except a slight attack of gonorrhœa in his youth. When in the army, during the war of the rebellion, he had a severe sunstroke, from the effects of which he was separated from his comrades several weeks, and because of which he was during the remainder of his service assigned to in-door hospital work. Since then, whenever exposed to heat, he has invariably suffered with intense headaches, dizziness, and other symptoms of the sequelæ of thermic fever.

He is described as a person accustomed to using alcohol moderately, but by no means intemperate in the use of intoxicating liquors. He is a moderate consumer of tobacco. He has a history of twenty-five years' use of opium.

In the autumn of 1883, while employed as an attendant in this institution, he was noticed to be suffering with fits of depression. He was sent away on a vacation with the hope of improvement under change of scene. In a few days he was returned to the institution as a patient, and the diagnosis of *acute suicidal melancholia* recorded. It seems that at this time he suffered from a delusion that he was the devil, although the history is rather vague, the book containing his record having been burned when the institution was destroyed in 1885. He remained in the house until the following February, when he was discharged recovered.

In the autumn following he returned, mildly depressed and still imagining himself one of the evil spirits undergoing torments. These fits of mental pain entirely altered his disposition, which formerly was of a happy buoyant nature.

In the early part of 1885, a complete change came over his delusion and disposition. As he describes it, he saw one night a clearly-defined vision. A number of horrible serpents came out upon his breath from himself and wriggled away, but a tall powerful man with the halo about him met them and slew every one. At the same time, the patient saw upon the ground before him a slip of paper which he picked up, and read this inscription: "Thou art the Son of the living God." A devout Catholic before, he now framed a new system of theology for himself, in which he figured as the carnal representation of the Divine Head. His argument was that in the beginning God had created the world, not from nothing, but from his own personal and material (!) self; that while man, His highest creation, was formed not only from His person but in His likeness, all else partook of His individuality, and held a share in His own eternal life. The patient's claim to supremacy he defended not only by the written statement he had seen in his vision, but by certain other to him indisputable proofs. Years before, some companions had tattooed on his arm his initials, P. J. O'F., and surrounded the letters by a vine and several roses. The letters now became to him the initials of his new title, "Patrick Jesus, Our Father," the roses were the roses of Sharon; the vine, the olive twig. Not that this interpretation was the one intended by his companions when they marked him for life, not that this use of his name was intended by the father who gave it to him, but according to his firm belief the whole train of circumstances was ordained from the first by the Divinity. So, too, to him certain scars upon his hands and feet and brow, the results of injuries well remembered by him and related without hesitation, were the visible proofs of his having suffered on the cross. The very name Patrick was additional evidence that he was the father, "*pater, qui in celum sit.*" To him, all being part of the One great and good, there could be nothing evil, no devil, no hell. To him in the theory of transmigration of souls there could be no impossibility, nothing degrading. All the children of the Father, there could be no need of worship, and himself that Father, he could not and did not care to expect the rest of the divine creation to bow down before him.

With this delusion he went to the asylum at Norristown after the burning of this institution, and had remained fixed in it ever since. He never speaks of it except when questioned, and although believing it himself, does not demand a like belief from others. In fact, at times he is willing to admit that there is a possibility of its being a delusion, but he never gives it up. He considers himself a sane man, and is such practically on all other points. He is not the subject of any hallucinations; on all other

subjects his intellect is unimpaired as far as our observations have gone. He has an excellent memory even for minutiae. His present disposition is cheerful and happy; he is entirely satisfied with his surroundings. His physical condition is good; he is a large, well-nourished man, with clear open countenance. His knee-jerk, however, is entirely absent. His pupils are unequal in size; the iridic reflex, although slow, is present. There is no evidence of change of sensibility or motility thus far.

## Clinical Notes.

### NOTE ON VOLUNTARY PASSIVE MOTION IN CASES OF PARALYSIS OF THE EXTENSORS OF THE FOREARM.

LEWIS D. MASON, M.D.,

ATTENDING PHYSICIAN TO HOSPITAL FOR NERVOUS AND MENTAL DISEASES.

All muscles in disuse atrophy, hence the importance, in cases of disease where rest of the part is imposed or compulsory, of adopting such measures as may arrest muscular atrophy until the part recovers its normal condition of motion. Electricity, massage, and passive motion, one or all of these may be employed with advantage, each case determining for itself the conditions under which these means can be used.

In cases of paralysis of the extensors of the forearm, lead palsy or dropped wrist (so-called), the inability of the patient to move his wrist is a source of marked mental depression. Otherwise in apparent good health, he is deprived of his only means of support, his hand is powerless, and he is as effectually crippled as if he had not any hand. The slow progress of his case still further increases his despondency. Now if by any means we can cause the patient to move freely his paralyzed wrist (paradoxical as the assertion may appear), we have made an impression not only for good on the mind of the patient, but we have also actually made an advance in the treatment of the case.

The method is a simple but effectual one. The patient is requested to supinate his forearm; the palm of the hand is thus turned upward, the back downward. At the same time, as the position of supination is somewhat tiresome, he is directed to support the affected arm just above the wrist with the opposite hand. He now flexes the wrist upon the forearm—this he can readily do of his own volition—he then, when the wrist is at extreme or moderate flexion, relaxes the flexors, and the hand falls back to its original position of extension, *gravity* being the factor that causes it to fall back. The position of flexion of the wrist is again assumed and again *gravity* causes the hand to fall back. Thus the patient practises passive motion whenever he chooses to do so or according to the direction of his medical attendant. We have thus by this simple method of *voluntary passive motion* in a partially paralyzed limb secured a valuable adjunct in the treatment of a class of cases oftentimes tedious in the extreme both to the physician as well as the patient, and in which we are glad to accept the slightest hint that may prove of service in the treatment.

## Periscope.

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### ANATOMY.

**Ueber die Verbindung der sensibeln Nerven mit dem Zwischenhirn.** The Central Tracts of the Sensory Cranial Nerves. L. EDINGER. (*Anatomischer Anzeiger*, No. 6, 1887, Separat Abdruck.)

Edinger has investigated the central tracts connecting the V., VIII., IX., and X. nerve nuclei with the tween-brain, in foetal specimens from cats, dogs, calves, rabbits, and apes. He is the first to follow the method of Flechsig into the domain of comparative anatomy, and the interesting results demonstrate the value of this method of investigation. Fibres which leave the nuclei of the sensory cranial nerves cross to the opposite side of the medulla or pons, forming part of the transverse fibres (bogenfasern) of these parts, and on reaching the lemniscus and formatio reticularis turn upward (cephalad) to pass to the brain. They decussate with their fellows in the raphé, usually near to the area occupied by the posterior longitudinal bundle. It is thus evident that the nuclei of the sensory nerves, like the nuclei of the posterior columns of the spinal cord (nuclei gracilis et cuneatus), are connected with the opposite side of the formatio reticularis and lemniscus by decussating fibres. And the existence of a sensory tract for the cranial nerves in the lateral portion of the formatio reticularis and lemniscus is thus determined. He claims that by this method tracts can be made out which cannot be distinguished from one another in human foetal brains, and the drawings which accompany the article demonstrate that the simple structure of the lower brains warrants this assertion. The introduction of this new method of investigation deserves to be noticed, as it promises as fruitful results as that of any hitherto employed. M. A. S.

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**The Topography of the Cerebral Cortex.** VICTOR HORSLEY. (*Amer. Jour. Med. Sci.*, April, 1887.)

In the absence of a comprehensive monograph on the relation between the external surface of the head and the various encephalic regions, Horsley records his personal experience, which has so far fortunately been equal to the exigencies of ten cases submitted to operation, in localizing brain lesions and determining the particular part of the skull and soft parts covering the focus of

disease. The sulci of the brain are to be regarded as landmarks of functional areas, but not as boundaries of them, evidence of this being found in the localization of the motor centres which lie on both sides of the fissure of Rolando. It is necessary, therefore, to find the position of certain convolutions as well as of the fissures and sulci. A clear account is given of the relations of the fissures, sulci, and convolutions which are concerned in motion. The results of a long series of experiments upon monkeys, conducted by Horsley and Beever, are made the ground for numerous interesting statements regarding the extent of the motor areas upon the cortex. The view is urged that in any given part of the cortex, as minute as can be examined experimentally, there is represented a definite movement or combination of movements of a definite segment or segments of one or both of the opposite limbs; and that secondary movements are due to the subsequent invasion by the discharge of nerve energy of those portions of the cortex which lie nearest to and are in close relation to parts stimulated. There is, therefore, an overlapping of the borders of various motor centres, or, in other words, the commingling of neighboring representations of movement. This view is in harmony with the views of Exner and Luciani, now widely accepted. And in the drawings given to show the location of the face, arm, and leg motor areas of the cortex, the necessity of distinguishing absolute from relative areas is implied.

The lower third of the ant. and post. central convolutions from the precentral sulcus to the interparietal sulcus constitutes the motor area of the face; but this is subdivided into an upper anterior part, governing the upper face and angle of the mouth, a lower anterior part, governing the vocal cords, and a lower posterior part, governing the lower face and floor of the mouth. The statement is made that the exact details of the representation of the movements of the face and throat have not yet been investigated experimentally, hence the facts given are new; but to the further statement that clinical observation has not filled up the blank we may justly take exception, as well as to the further statement that clinical observation of the effects of disease furnishes but barren results. For the case of Amidon, of a small lesion in the facial area producing spasm of the upper lip and cheek, and the case of Krause, of localized spasm and paralysis of the vocal cord produced by a small lesion in the facial area, had demonstrated, at least three years ago, that such a subdivision of the general facial area was possible.

The middle third of the anterior and posterior central convolutions governs the upper limb, but its motor area also extends into the middle frontal gyrus, where it is blended with that of the head and neck, and into the superior frontal gyrus, where it is blended with that of the leg. In this area the shoulder is centred in its upper part, the elbow next below and posteriorly, the wrist next below and anteriorly, the fingers

next below and anteriorly, the thumb lowest and posteriorly. Viewing the movements of the limb as a whole, he finds that there is hardly one in which the elbow and wrist do not take part, while the wrist and elbow are rarely moved alone. Hence absolute centres for their movement are not extensive, but relative centres are quite widely distributed. He claims that the subdivision of the motor area for the arm is confirmed by cases of cortical tumor which he has operated upon, the beginning of the spasm in each case being different. In one case the fit began with flexion of the shoulder, and the tumor was on the upper part of the arm area. In another the fit began in the thumb and the tumor was found in the lower part of the arm area.

The motor area for the lower limb is very extensive, including the posterior sixth of the superior frontal gyrus, the upper third of the central convolutions, the paracentral lobule, and the superior parietal lobule as far back as the parieto-occipital fissure. The anterior part of this region governs combined motions of the leg and arm, the middle part of the leg alone, the great toe being represented in the paracentral lobule. Further subdivision of this region is reserved for another paper. But cases are cited which prove that such a subdivision is probable, spasm and paralysis limited to the great toe having been in two cases the early symptom of lesion in the paracentral lobule. The movements of the head and neck, with that of conjugate deviation of the eyes, are governed by the area lying in the posterior part of the three frontal convolutions—a conclusion which confirms the statement of Munk, reached six years ago, to which, however, no allusion is made.

Having thus determined the exact location of the motor areas of the cortex, the topographical relations of these to the skull is considered. The fissure of Rolando is first located, according to Thane's method. The length of the middle line of the head, from the root of the nose to the occipital protuberance, is taken and halved. One-half inch behind the centre point of this line, the upper end of the fissure of Rolando is found in adults. The angle made by the fissure with the middle line is sixty-seven degrees. A strip of flexible iron having an arm attached at its middle, the arm making an angle of  $67^{\circ}$  with the strip, is used as a means of measurement, and when the strip is laid upon the middle line of the head and the junction of the arm and strip placed over the theoretical situation of the upper end of the fissure of Rolando, the arm lies over the fissure. But as the fissure of Rolando bends slightly backward in its lower third, the arm of the instrument indicates only the upper two-thirds of the fissure. The fissure of Sylvius is next located. It commences at the pterion, and passes upward and backward as far as the highest point of the squamoparietal suture, whence it curves slightly upward toward the centre of the parietal eminence which it nearly reaches. The pterion is half-way between the stephanion and the upper border of the



zygoma, the measurement being taken along a line drawn vertically to the zygoma from the stephanion. The stephanion is the point where the temporal ridge crosses the coronal suture, both of which can be readily made out by steadily pressing the scalp with the thumb over their supposed sites. If the coronal suture cannot be felt, there can be felt a rounded ridge bounded by two grooves, and the suture lies in this ridge. The highest point of the squamoparietal suture is under the temporal muscle in a vertical line drawn in front of the articulation of the lower jaw, being at the point at about the junction of the upper and middle thirds of the distance between the ridge of the temporal muscle and the upper border of the zygoma. The anterior branch of the fissure of Sylvius runs upward and forward from the pterion, continuing, as it were, the line of the sphenoido-squamous suture, but commencing one or two millimetres in front of it. The precentral sulcus runs parallel to and just behind the coronal suture, and reaches to about the centre of the fissure of Rolando; from it diverges the inferior frontal sulcus about opposite to the superior temporal ridge. The superior frontal sulcus commences in the ascending frontal convolution about midway between the fissure of Rolando and a line continued upward in the line of the precentral sulcus. The interparietal sulcus, which forms the posterior boundary of the motor area, can be located after the position of the fissures of Sylvius and Rolando are known; for it begins opposite the knee-like bend in the fissure of Rolando, and turns backward just below the horizontal level of the superior frontal sulcus. Here it lies midway between the fissure of Rolando and the centre of the parietal eminence. Further up, as it passes backward, it lies midway between the longitudinal fissure and the centre of the parietal eminence. The parieto-occipital fissure lies just in front of the lambdoid suture. Having found the fissures and sulci, the situation of the convolutions can be readily determined. M. A. S.

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PHYSIOLOGY (INCLUDING PHYSIOLOGICAL PSYCHOLOGY).

**The Time Taken up by Cerebral Operations.** (*Mind*, April, July, and October, 1886.)

Dr. J. M. Cattell, in the psychological laboratory at Leipsic (Wundt's), has made a re-determination of the reaction-time for various mental processes, and claims to have used improved methods of recording, and to have eliminated errors that have formerly been made in making out the averages. The times were recorded by a Hipp chronoscope, which was controlled by an instrument devised by Cattell and called the gravity-chronoscope, by means of which the error (sometimes amounting to one-tenth sec.) due to the time consumed in magnetizing and demagnetizing the electro-magnet of the Hipp instrument is eliminated. For obtaining the correct average, he has used a different method from that used by Exner, Merkel, and others. A reaction may vary so from the average that the whole series will have a false value. Exner

simply ignored reactions that seemed too long or too short, but Cattell criticises this by saying that the experimenter thinks he has found the proper worth, and then almost unconsciously leaves out of his reckoning the reactions which would invalidate it.

C. has taken a series of 13 or 26 reactions, calculated the average, and the variation of each reaction from the average. Then he has dropped the reaction having the largest variation; the average of the 12 or 24 reactions remaining has been calculated anew, and the reactions varying most from this average again dropped. In this way the 3 or 6 worst reactions have been dropped, and the 10 or 20 best reactions remain with the variations of each of these from the average. In tabulating the results, .001 sec. has been taken as a unit, and he uses the symbol  $\sigma$  to represent this unit, analogous to  $\mu = .001$  mm.

The following table is a summary of the results for the two observers, Berger and Cattell.

	B.	C.
Reaction-time for light.....	150	150
Perception-time for light.....	30	50
"    " a color.....	90	100
"    " a picture.....	100	100
"    " a letter.....	120	120
"    " a (short) word.....	120	130
Will-time for colors.....	280	400
"    " pictures.....	250	280
"    " letters.....	140	170
"    " words.....	100	110

No explanation of the reaction-time is necessary, as this means the same to all observers, but an analysis of what C. considers to make up the perception-time and will-time is of interest. He defines the perception-time as the interval between sensation and perception (or between indefinite and definite perception), the time passing after the impression has reached consciousness before it is distinguished. Wundt obtained his results by letting the subject react as quickly as possible in one series of experiments, and in a second series not to react until he had distinguished the impression, the difference of the time in the two series giving the perception-time for the impression. Cattell was not able to get results by this method. Donders, von Kries, Auerbach, and others thought that if the subject reacts on one of two impressions and makes no motion when the other occurs, only a perception has been added to the simple reaction. C. claims, and with justice, that this is not the case, it being necessary, after the impression has been distinguished, to decide between making a motion and not making it. Cattell assumes that the changes do not penetrate into the cortex at all when a simple reaction is made; but when

lights of two different colors are used (say red and blue), and the subject may only lift his hand if the light is blue, the motor impulse cannot be sent to the hand until the subject knows that the light is blue; the motor impulse must therefore travel to the cortex and excite changes there, causing in consciousness the sensation or perception of a blue light; this gives a perception-time the additional time necessary for a nervous impulse to be prepared and sent to a motor centre, and discharge as a motor impulse gives the will-time. The point is worth emphasizing, and C. does not think it possible to add a perception to the reaction without also adding a will-act, agreeing with Wundt, but differing from Donders, von Kries, and others. C. changes the nature of the perception without altering the will-time, and thereby claims to get, with considerable thoroughness, the length of the perception-time.

C. assumes that the time of the centripetal and centrifugal progress through the brain (or the perception-time and will-time) is about the same, and that the time used in the cortex is about equally divided between the perception of the light and the preparation of the motor impulse, and so by dividing into two parts, the remainder obtained by subtracting the simple reaction-time from the whole time, including both discernment and choice, estimates the perception-time for light, of B. at 0.03 sec., and of himself at 0.050. With regard to these results, we can only say that the matter is still *sub judice*, and that we cannot yet, with confidence, assign to each element of the psycho-physical time its true value.

WILLIAM NOYES.

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**On the Reaction-Time for Auditory Impressions in Hysterical Subjects in the Different States of Hypnotism and especially in Echolalia.** (*Revue Philosophique*, April, 1886.)

In these experiments, reported to the *Société de Psychologie Physiologique*, the subject held a telephone against her ear, and to her chin was attached an apparatus by which an electric current made a record on a Marcy tambour every time she pronounced the word "toc;" the telephone was also in a circuit that made a record on the same cylinder with a Deprez signal; when the electric contact was made it produced at the same time a noise in the telephone and made a signal on the tambour. The hysterical subject said "toc" each time she heard a noise in the telephone, and as quickly as possible, thus giving the reaction time for this patient for auditory impressions. In the *waking state* the reaction-time was thirty-nine hundredths of a second. In the condition of *somnambulism* the reaction-time was not more than thirty-three hundredths of a second. The subject was then placed in a condition in which she exhibited the phenomena of *echolalia*, that is, during *somnambulism* one of the experimenters placed his hand on the top of her head and she repeated faithfully all the sounds that came to her ear. She reproduced the noise made in the tele-

phone by a sound very much like the word "toc." Now in this case the reaction-time was only thirty-one hundredths of a second, or three hundredths less than in the simple somnambulistic state. The results are interesting as showing that in echolalia the will appears to be completely absent, and this interval of three hundredths of a second measures the duration of the voluntary psychic operation which was suppressed by the appearance of the echolalia. The original communication is not given, but it is difficult to see why the *will-time* is not two hundredths of a second rather than three as stated by the reporters MM. Marie and Azoulay.

WILLIAM NOYES.

### The Anatomy and Physiology of Touch.

It is just about three years since the study of the dermal sensations was enriched by the epoch-making discovery of separate points for the reception of cold and of warm sensations. The names associated with this discovery are Magnus Blix, of Upsala, Alfred Goldscheider, of Berlin, and H. H. Donaldson of Johns Hopkins University. Dr. Goldscheider has, however, carried the work on in greater detail than any one else, and the papers published within the last year would make quite a respectable volume. He has just added to these an account of the microscopic appearances of sections of skin containing the temperature and other points. (*Archiv für Anatomie und Physiologie*, Supplement, 1886.)

Dr. Goldscheider distinguishes three kinds of points on the skin: cold-points, heat-points, and pressure-points; each of these kinds of points are arranged in chains running in a somewhat curved manner, the chains generally radiating from certain points of the skin. These points of radiation are apt to coincide with the insertion of hairs.<sup>1</sup> The cold- and heat-points cannot arouse pressure or pain sensations, but when stimulated give rise to temperature sensations only. On the pressure-points pressure sensations are aroused and the prick of a needle gives continued pain. On intermediate points pressure and pain are felt in a slight measure. The points do not all react with equal intensity.

Starting with these physiological facts, the problem is to find their anatomical basis. The law of the specific energy of nerves has been justified, *i. e.*, every nerve reacts in one and but one way, no matter how it is excited. The same nerve fibre cannot give rise to both temperature and pressure-sensations, but there must be entirely separate fibres for each. The anatomical proof of this prediction of physiology is a very delicate task. The skin abounds in all sorts of nerve fibres, and to distinguish one kind from another seems almost a hopeless task. It is easy to trace the endings of the optic or the auditory nerves, because no other fibres occur

<sup>1</sup> It should be stated that in his arrangement of the points and in his views on the nature and existence of the pressure-points, Goldscheider differs very much from the other observers.

in their immediate vicinity. The most promising method of differentiating the nerve endings in the skin is to locate typical heat-, cold- and pressure-points exactly, then cut them out from the living human skin and prepare sections for microscopic study. Dr. Donaldson tried this method on himself, but with a negative result. Dr. Goldscheider has refined the method of excision and preparation and offers results which, though not final or complete, are welcome as they are suggestive.

The point to be excised was marked accurately with indelible ink and carefully cut out. In cutting out a heat-point, there was a sudden intense burning sensation; a cold feeling was aroused in cutting out a cold-point; and in excising a pressure-point the pain was much more severe and constant than in the case of temperature-points. This is analogous to the vivid light caused by operations on the optic nerve and shows the universality of the law of specific nerve energy. It also shows the insensibility to pain of the temperature-points; a needle can be run into these without causing pain. As to the microscopic appearances the points of importance are these: (1) In each case nerve fibres are found *directly* under the marked sensitive points, thus showing the anatomical basis of these sensations. (2) The fibres here have an upward course, and as far as they can be traced end freely, or perhaps in knots amongst the limiting line of cells of the cutis. (3) No anatomical distinction between the heat- and cold-points has as yet been found. (4) The temperature nerve fibres and the pressure fibres can be somewhat distinguished by the manner of their distribution and their relation to the capillaries. (5) There is no network of fibres, but each runs separately throughout its course.

These results, meagre as they are, nevertheless suggest some important considerations. One would expect to find terminal organs of some kind to serve for the reception of the dermal stimulus; this expectation is not realized. Their absence suggests that the explanation of the mode of action of the nerve fibres must be sought in their terminal distribution, that is, in the grouping of the endings. The physiological unit may be anatomically complex. At a pressure-point the skin possesses a number of nerve fibres which are spread out flat in the sub-epithelial cutical layer and by numerous branches supply a relatively large piece of dermal surface. The magnification of the surface supplied by the nerve fibre may serve to give discontinuous stimuli the effect of continuity, and thus serve the function of an end organ. At a temperature-point, a number of fine nerve fibres are crowded together in the immediate vicinity of blood-vessels. These fibres must be supposed sensitive to temperature changes; and, as before, a complex system of fibres serves as the end organ. If but one fibre supplied each point, it would take much more intense stimulation to produce sensation. Perhaps the happiest suggestion to which the absence of terminal cells leads Dr. Goldscheider is that the Meissner touch-corpuscles

are merely protective organs. These are found on such parts of the body (finger-tips, palms, sole) as are used for fine sensibility and yet are subject to violent pressure and injury. The corpuscle simply protects the nerve endings and thus allows a fine sensibility to be developed.

The time for writing the physiology of dermal sensations has not yet come, but the discoveries of Goldscheider and others give promise that the initial chapters of that book have been or are about to be written.

JASTROW.

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#### PATHOLOGY.

**Removal of a Large Sarcoma, Causing Hemianopsia, from the Occipital Lobe.**<sup>1</sup> By W. R. BIRDSALL, and by ROBERT F. WEIR. (*Medical News*, April 16th, 1887.)

The following history, herewith reported conjointly by Dr. Weir and Dr. Birdsall, is that of a patient who during life presented left hemianopsia, optic neuritis, and certain disturbances in locomotion, from which the diagnosis of tumor of the right occipital lobe was made by several physicians, who also concurred in advising an operation attempting its removal, a fatal result appearing otherwise inevitable. As predicted, the tumor was found in the region described. It was removed in the manner to be stated by Dr. Weir, death resulting subsequently.

Male, æt. forty-two, a Hebrew, native of Poland, came under B.'s observation October 16th, 1886. Until the summer of 1885, he had always been healthy, and denied ever having had any form of venereal disease, or injury to the head. In August, 1885, after a sea-bath, he observed, for the first time, unsteadiness of gait, and had a severe attack of vomiting. Soon after diplopia for distance and increased awkwardness in walking were observed, and about the same time a disagreeable sensation, akin to numbness, in the right leg, hand, and shoulder, but not in the face. This and the diplopia were transitory. Headache, usually frontal, was present occasionally, but was never severe. Vertigo, or tendency in a definite direction, was not noticed at this time. No other sensory, motor, or visceral symptoms appeared. He was observed to miss articles when told to pick them up. This was probably due to the diplopia.

Oct. 7th, 1885. Dr. Seguin was the first to recognize in the patient the important localizing symptom, hemianopsia, and to make the correct diagnosis, and also to have seen him before and during the development of the optic neuritis, and at a recurrence of the diplopia.

"Examination: Eye muscles normal (no diplopia with red glass). Left pupil a trifle wider than right; both active; fundus normal. Has left lateral hemianopsia, vertical line passing a little

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<sup>1</sup> In view of the interest attached to this case, the report is reproduced almost verbatim and at more than ordinary length.—ED.

to left of fixation point. No paresis of face, tongue, or limbs ; no anæsthesia ; patellar reflexes normal ; walk is somewhat staggering with decided tendency to right.

"29th. Diplopia has recurred ; left externus weak ; hemianopsia the same. No hemiopic pupillary reaction. Grasp : R.,  $42^{\circ}$  ; L.  $30^{\circ}$ .

"Nov. 7th. Beginning neuro-retinitis discovered. This was verified ten days later by Dr. Gruening, who had already seen the patient for hemianopsia, and determined the extent of his visual field. Both externi paretic (this passed away later). His wife states that drowsiness in the daytime has been a marked symptom from the first ; also difficulty in rising from the chair."

Diagnosis made November, 1885 : Tumor of mesial aspect of right occipital lobe, involving primarily the cuneus, extending downward toward the tentorium cerebelli, and perhaps also upward toward the paracentral lobule (leg centre). During this period the patient was treated with large and increasing doses of potassium iodide.

From January to July, 1886, he was under the care of the late Dr. McBride. During the early months of this period his diplopia disappeared and never returned. His gait improved somewhat, though he had two attacks in which he suddenly, while walking on the street, felt that he could proceed no further, and at the same time had a strong tendency toward the left, and actually staggered to the left so that his wife was obliged to support him. After resting a few minutes he was able to walk again. No other symptom of importance could be elicited from the patient or his wife covering this period. In July, patient took a trip to California, and returned in September, in about the same condition, from which time on his difficulty in rising, standing, and walking, together with drowsiness, rapidly increased.

At the time of examination in October, 1886, left lateral hemianopsia was present. The ophthalmoscope revealed double optic neuritis, most marked in the left eye. Iris active to light and accommodation, the left pupil somewhat larger than the right ; no diplopia ; no ocular paresis evident. Smell, taste, hearing, and speech normal ; no word deafness or word blindness ; no anæsthesia, analgesia, or disturbance of temperature sense in any part of body. No paresis in muscles of face, trunk, or extremities. No tremor of tongue, face, or extremities. In testing the patient for ataxia by touching finger to the nose with eyes closed, his movements were clumsy rather than truly ataxic, at times being accurate, at others wide of the mark ; this was particularly noticeable with the left hand. A similar condition was noticed in the lower extremities, also more marked on the left. His gait was slow and uncertain. He was unable to find words to express the defects which he felt ; denying that it was muscular weakness, numbness, pain, tremor, stiffness, or vertigo, which caused his cautious yet awkward movements. Uncertainty of control seemed to be the

best term for it. He referred this chiefly to the right hip, thigh, and knee. His patellar tendon reflexes were rather active and about equal. In walking, he would frequently hit objects to his left, probably on account of the limitation of the visual field upon that side, and when seated at the table he would turn to the left, bringing a half profile view of the body and face to a person directly opposite. This was, probably, also due to the same cause, being an attempt to bring his limited field fully to the front of the table. There was no tenderness to pressure or pain on percussion of any part of the head. Frequent expectoration of a viscid saliva was an increasing symptom during the last few months of his life.

*Diagnosis.*—The symptom left hemianopsia could only be accounted for by a destructive lesion in the neighborhood of the gyrus cuneus of the right occipital lobe. The locomotory disturbances appeared to be due to the pressure effects of a tumor on structures below the tentorium, this implying a growth of considerable size.

No new symptoms developed, except that the right patellar tendon reflex became somewhat greater than the left. His disturbance of equilibrium continued to increase in a very irregular manner. On some days he would stand and walk quite well, on others he would suddenly stagger in walking, or fall over to one side when seated, usually forward and to the right, frequently with a twisting tendency of the body to the left. His steps became short, the feet being barely lifted from the floor, and with movements slow and cautious. Yet he could see well, reading the daily papers up to the last. On some days he was dull and listless, though never in a stupor; on others, was bright and talkative. His intellect was not impaired, and his family observed no change in his character or disposition. His sleep was natural, and hallucinations were never observed. He was extremely uncomfortable mentally, not from his visual defects, to which he attached little importance, but from his difficulties in locomotion, and disagreeable sensations.

Treatment with potassium iodide, which had been discontinued for some months, was resumed.

Drs. Seguin, Birdsall, Spitzka, and Jane May advised operation.

The characteristic feature of all his symptoms, except the hemianopsia and the optic neuritis, was their vacillating and intermittent nature, even to the oculo-motor disturbance, indicating pressure effects, or circulatory disturbance, rather than direct destructive action of the tumor.

The operation was performed March 9th, 1887, at the New York Hospital, whither the patient had been sent for more perfect control, and on account of the better antiseptic conditions there present. A dose of Hunyadi water was administered to move the bowels the morning of the operation. The head was also shaved, and the scalp washed with green soap and water, and then with



ether, and subsequently covered for several hours with carbolic cloths, wrung out of 1 : 30 solution and by gutta-percha tissue, all secured *in situ* by a bandage.

At 3 P.M., in the presence of Drs. Birdsall and Seguin as neurological counsel, and of Drs. Bull, Markoe, Abbe, Hamilton, C. T. Bull, Olcott, Starr, Dana, Sachs, and others, the operation was undertaken. The bregma, Rolanderic, and median lines having been marked out, and the occipital protuberance with some difficulty identified, and after the patient had had a hypodermatic injection of a quarter of a grain of morphia, and had been etherized, a U-shaped flap, three inches long and three wide, with base upward, was made under a carbolic spray, 1 : 30, so as to straddle irregularly the median line in its lesser part, the greater part being over the right posterior cerebral lobe. The bleeding was free from this, and from the thick periosteum, and also from the skull itself. At one inch above the occipital protuberance, and the same distance from the median line—in other words, beyond the limits of both the longitudinal and lateral sinuses—the circumference of a one-inch trephine was placed, and the bone, which was rather thin, cut through. A second button was removed immediately above the first, and the intervening bridge gnawed away by a rongeur forceps. The edge of this resulting aperture was further enlarged by taking away externally the cranium with the same instrument until an oval opening, measuring two and three-quarters by two and a quarter inches, was made. The dura mater, non-pulsating, rose tensely in the space, and was of a deeper hue than normal. This membrane was divided for two-thirds of the extent of the bone opening, its retained attachment being toward the median line, so as to avoid encroaching on the longitudinal sinus. As it was cut and turned back, the brain—or what was at first taken for brain, but was immediately recognized as the tumor—at once rose into the bony opening. It was of a purplish-red color, like kidney structure, and was covered over by a thin cellular tissue, with large veins ramifying in it. With a director and the edge of a spoon handle, a thin, yellowish layer of flattened-out, expanded brain tissue was loosened from the tumor on its outer side, and in this direction the enucleation was accomplished to a depth of nearly an inch. Similarly proceeding, but without seeing any further brain substance, the tumor was loosened easily on all sides. Additional room was obtained for manipulation by cutting away freely of the cranium externally, but all was insufficient to obtain access to the outlying edges and base of the growth. The tumor was, therefore, incised, and some of its softened, granular, and fatty-looking contents forced out. This somewhat diminished its size, and enabled the forefinger to be passed between the cranium and tumor, and by its aid the delicate cellular attachments that held the mass in place were felt to yield easily, and enucleation became possible, and the base finally reached. By now drawing the finger gently, but firmly, toward the cranial opening, the tumor was torn nearly

completely in two, and its outer half lifted out, and then the inner part, with the help of the finger-end and nail, separated from the falx and withdrawn.

Inspection of it showed that it had been entirely removed, and that its probable attachment was toward the posterior border of the falx. A good deal of venous bleeding took place from the huge cavity left by the removal of the tumor. This was stopped with four sponges, and temporary pressure in this way resorted to. After a few minutes they were removed, and the cavity inspected by the light of a small electric lamp, which showed the immense compression of brain tissue that had taken place, the falx being crowded over toward the left beyond the median line, and the tentorium depressed to a horizontal line. The tumor itself told the story better, for it, as was afterward learned, weighed one hundred and forty grams, or five and a quarter ounces, and measured three and a quarter inches by two and three-quarters, and was two and a half inches thick. Its greater circumference was eight and a half inches, and its lesser seven inches.

As the hollowed-out brain was lifted up by a retractor, two bleeding points were seen, one being in the region of the straight sinus, though not free enough for that vein, and probably belonging to the pedicle of the growth, and the other apparently was arterial, and possibly from a terminal branch of the posterior cerebral artery. The flow from each was readily checked by direct pressure, and it was determined, on consultation, to control them by packing this cavity with iodoform gauze of five-per-cent strength. This was done not too strongly, it being assumed that the released brain would also contribute additional pressure, and the ends of the strips of gauze subsequently were, for easy extraction, allowed to emerge from the lower angle of the scalp wound. The dura mater was partly united over the gauze by several loose sutures (instead of being closely brought together, as had been done in another case), and the scalp wound closed with catgut sutures, a rubber drainage tube being introduced under the skin up to the skull opening. Over these, sublimated and iodoformed peat bags with sublimated loose compressors of gauze and absorbent cotton were secured with gauze bandages, and the patient put to bed.

The operation was well borne until the final enucleation took place, when the pulse decidedly fell, apparently from the loss of blood, which was then suddenly augmented, and which amounted, in the whole operation, to some ten or twelve ounces—the oozing being persistent from the scalp and diploë, and difficult to control entirely by ligatures, clamps, and finger pressure. By bandaging the limbs and by the administration of whiskey subcutaneously his condition improved, and at the close of the surgical work he was in a fair condition. Pulse 132, but regular and of good volume. Slight diverging strabismus was, however, noticed in the left eye. He was ordered stimulants by the skin and rectum.

Hot bottles, duly protected, were placed in the bed, and his head kept low, and enemata of whiskey,  $\frac{5}{8}$  i., and milk were ordered every two hours, with stimulants, hypodermatically, if required.

The patient came out of the ether quickly, and showed considerable restlessness, moving all his limbs and having proper voice. By 7.30 P.M., two hours after the operation, the pulse had become slower, 120, but gradually weaker, and the dressings were stained with blood which had soaked through them at one point. This deterioration of the pulse continued until I saw him with Dr. Birdsall, at about 10 o'clock, when he was found very restless, and with an extremely weak pulse, and increasing marks of blood soakage in the dressings. The patient, however, was conscious, though somewhat dull. A salt transfusion (common salt, 93 grains, carb. sodæ, 16 grains, to one quart of filtered water, to which solution three per cent of sugar is added, as suggested by Landerer) of nearly two quarts was slowly injected into the median basilic vein at the right elbow with immediate improvement in the pulse and consciousness. He became also more quiet, and could answer questions, and put out his tongue fairly straight. He, however, showed signs of slight paralysis of the ocular branch of the seventh nerve on the left side, and also had decided divergent squint of the left eye. His hemianopsia was tested by Dr. Birdsall, and found to be unchanged.

As it was evident that part of his deterioration was due to a loss of blood, it became imperative to see if its continuance could be arrested. The dressings were, therefore, quickly removed, and the flaps of the scalp freed by cutting the stitches and raised, when blood was seen to escape in a small stream through the tube from the brain cavity. At first it was intended to remove the packing, and to secure the bleeding points by clamps; but his pulse, which had been raised by the transfusion, suddenly gave out, or so nearly so as to cause me to abandon the idea of resorting to any procedure of length, and to content myself, with Dr. Birdsall's approval, of further crowding in additional iodoform gauze toward the supposed source of hemorrhage. This was done, the flaps replaced, but not resutured, and dressings reapplied. Symptoms of stupor quickly came on, and fearing lest the pressure might cause this, the dressings were loosened by cutting, but the patient's condition continued alarming, and his pulse became absent at the wrist, but was restored by a second transfusion. While the circulation was thus improved, the other symptoms were not similarly affected. The transfusion was kept up experimentally, though the patient's condition was hopeless, and by its means the heart was kept acting till 2 o'clock A.M., when he died.

No autopsy was allowed on account of religious scruples, but on removing the packing of iodoform after death, in the lower and anterior part of the cavity was seen quite a large collection of coagulated blood. The tumor was reported by the pathologist of the hospital, Dr. Peabody, to be a spindle-celled sarcoma with a few round cells sparsely found in it, and not to be very vascular.

REMARKS BY DR. WEIR.—There are several surgical points of interest in connection with the forgoing case that may be cursorily dwelt upon. The most important, because it largely entered into the cause of death, was the erroneous method adopted of arresting the hemorrhage. I had previously encountered lacerated vessels in the substances of the brain, the first time in 1882, and twice since then, and had secured them by ligature or by torsion, but none of these were at a greater depth than an inch from the surface. From the effects of sponge pressure, I was led to believe in the present case that the openings in the blood-vessels could be easily controlled, in which idea I was mistaken; the bleeding was also probably favored by the headlow position which his shocked condition induced me to direct. It would have been more correct perhaps to have tried cautiously to elevate his head, and in this way to diminish the blood pressure. On a review of the case, however, I believe it would have been better surgery, and in another instance I would so act, to control the bleeding at once from vessels too deeply placed for a ligature, by means of clamp forceps which might protrude through an opening in the flap, and be removed after a period of twenty-four or forty-eight hours, as is done by Richolot's forceps in the vaginal removal of the uterus.

The size of the tumor, it is hardly necessary to state, exceeded anticipation, those usually encountered being smaller, although one has recently been reported by Horsley which weighed four ounces, and produced hemiplegia and coma at the time of the operation. In the present operation, though the size of the skull opening was fully two and three-quarters by two and one-quarter inches, further bone room would have allowed an easier extraction of the growth. This enlargement was most desirable toward the median line, and would have been resorted to without much hesitation had the attempt at enucleation failed, for sundry experiences of injuries over the longitudinal and lateral sinuses, together with those obtained in the cadaver, had convinced me that the skull over such a sinus can be removed without opening it, and without giving rise to any uncontrollable bleeding or subsequent risk. In the rehearsals made for this particular case, which were conducted on the possibility of the growth projecting from the inner side of the cuneus against the falx, as was seen in one of Dr. Seguin's cases, it was ascertained that after the bone was gnawed away over the longitudinal sinus, that the dural flap, whose attached base was toward the sinus, could be so pulled upon as to expose fairly the median plane of the brain, aided by a spatula lightly pressing the latter outward. The same procedure could be applied to the inferior surface in respect to the lateral sinus, so as to expose to a considerable depth the tentorium. Such an examination was conducted in a patient whom I shall present in a few moments to illustrate another point, and in whom a frontal lobe was largely opened up to view for the relief of traumatic epilepsy of thirty-five years' duration. The lateral sinus, I may also remark, has been

exposed by others besides myself, viz., by Schondorff, Lucae, and by Knapp, to a varying extent, without mishap.

The size of the skull opening, therefore, should be large, and Horsley advises the use of a two-inch trephine, and makes two openings with this instrument, connecting them with a saw and cutting forceps. The apprehension that this large vacuity in the calvaria would subsequently expose the patient to the risk of easily inflicted cerebral injury, is not so great as imagined, and can be greatly lessened by resorting to the expedient first practised elsewhere in the body by Macewen, of employing bone grafts, and by sprinkling, as Horsley does, over the dura after its edges have been sewn together, the chopped-up disk of bone, which is to be carefully kept warm till the completion of the operation.

A further step in this direction has been made by Poncet, who has shown that pieces one-third of an inch long and one-sixth of an inch wide, can be similarly used. I recently ventured, in the case of trephining for epilepsy which was just mentioned, after exposing the brain and dividing an adhesion extending between the pia and dura mater, through an opening nearly two and a half by three inches, to replace, after closing the dural opening, the two one-inch disks of bone which had been removed by the trephine. These had been wrapped in a towel wrung out of warm carbolic solution, which in its turn was then placed in a jar immersed in warm water. The operation lasted fully half an hour before the bones were put back. It is now seven weeks since the operation, and you will perceive, in the patient who is now submitted to your inspection, that the wound is all healed, save at one point over the eyebrow, where an opening was made recently downward through an obstructed suppurating frontal sinus to the nose, to permit drainage, and that no communication leads to the circles of bone, which can be felt above the point, solid, resisting, and painless. Later still, Dr. McBurney, at St. Luke's Hospital, has repeated this procedure, after an exploratory operation, for brain disease. This plan, if corroborated by further experience, will relieve our minds of the objections held to large openings in the skull, and will facilitate greatly bolder surgical explorations.

Up to the present time the opening of the skull for the extraction of a contained tumor has been resorted to eight times, once by Bennett and Godlee in 1884, three times by Horsley in 1886, which, with the one above narrated, make up the five cases of removal of a tumor, the result of which in two of Horsley's is yet unknown, but presumably it was a successful one. Of the three other cases, in one by Hirschfelder and Morse in 1886, the tumor was found, but only a part was removed, the patient dying shortly afterward from suppurative encephalitis; in the two remaining cases no tumor was found, though in the one operated on by me, and reported at length in *The Medical News* for March 5th, 1887, at the post-mortem, two and a half months later, a tumor was found pressing upon the cerebellum and spinal cord.

The last case is the one reported by Dr. G. M. Hammond, and made the subject of the paper succeeding this, to be read before this Society, in which the search nearly succeeded, as was shown afterward at an autopsy. The cause of the symptoms was the presence of three cysts adjacent one to another, and thought to be of hydatid origin.

REMARKS BY DR. BIRDSALL.—In concluding, I may be allowed a few remarks concerning the tumor which was found. Owing to its large size, so much of the occipital lobe was compressed by it that the case is of little value for the purpose of determining the limitation of the visual area in the occipital lobe. The growth was a sarcoma, originating in the meningeal structures, and producing destruction of the cerebral tissues by pressure alone; no part of it was infiltrated into the cortex. The absence of severe headache in this case should be noted, as it is usually a prominent symptom of tumor involving the meninges.

That convolutions may be reduced to the thinness of paper by such a process is well known, and in this case the apex of the occipital lobe was literally crushed between the tumor and the cranium, while the more frontal portions were compressed in that direction. The parts beneath the tentorium were also compressed, as the symptoms during life led us to infer. The remarkable feature of cases with so large a tumor is not so much that they give rise to localization symptoms, as that they exhibit so few.

One of the most important lessons that the study of cerebral tumors teaches is that growths remaining limited to meninges may attain a large size before disturbing the function of neighboring parts of the brain, frequently giving rise to less marked symptoms than very small growths, which infiltrate the cortex. In the deeper conducting tracts of the brain, where fibres run more in parallel courses, growths may attain large size without producing much irritation or destruction, by slowly pushing the fibres aside; this gradual expansion could not go on in the felt-like mass of fibres in the cortex without destructive action resulting. Thus, in one of my reported cases,<sup>1</sup> a sarcoma the size of a hazelnut displaced the cortex of the arm area, producing spasm and paresis of the arm, while a similar growth under the same area of the opposite hemisphere, but a few lines deeper yet not reaching the cortex, gave rise to no symptoms whatever. Again, when tumors destroy by pressure, the softer mass of the growth may injure less than the rigid walls of bone against which the cerebral tissue is compressed; so that regions away from the tumor may give signs of impairment before those in contact with the tumor. These are some of the contingencies (and there are others) which will probably always constitute obstacles to the correct localization of tumors, as guides to surgical operations for their removal.

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<sup>1</sup> Arch. Med., vol. ix., No. 3, 1883.

## Reviews and Bibliographical Notes.

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### **A System of Practical Medicine by American Authors.**

Edited by WM. PEPPER, M.D., LL.D. Volume V. Diseases of the Nervous System. 8vo, pp. 1,326. Philadelphia : Lea Brothers and Co., 1886.

In spite of the fact that the volume before us is the largest single volume on diseases of the nervous system that has ever been published, it is not large enough to contain all the articles on nervous diseases that have been contributed to this System, for the contributions on myalgia, progressive muscular atrophy, and pseudo-hypertrophic paralysis have been placed in the previous volume. The volume before us forms a fitting conclusion to the great System of Medicine which in so short a time has won so high a position in medical literature, and, in itself, it is a contribution to our knowledge of nervous diseases of which American neurologists may well be proud.

One of the striking features of the volume before us is the amount of individual investigation and personal experience that the contributions represent—a feature which might, of course, be expected from the character of the contributors, but which becomes more noticeable on viewing the entire work as a whole. While almost every article shows the results of wide reading, hardly one of them degenerates into a summary of other men's work.

Divided as the work has been among so many contributors—twenty-nine in all—we miss, of course, the unity and system which render the exhaustive treatises of Erb and Kussmaul so valuable in the great German *Handbuch*. This fragmentary character of the work before us is due in part to the defective classification of diseases adopted—nominally that of the Royal College of Physicians, but practically the same chaotic arrangement as is employed in Reynolds' System of Medicine. To this imperfect and incomplete classification we owe, perhaps, the entire omission of certain subjects—for example, arsenical paralysis, tetany, subacute poliomyelitis, compression of the cord, the acute diseases of the medulla, and, worst of all, an account of the individual forms of peripheral spasm and paralysis. Another unpleasant, but probably unavoidable feature is the lack of unity between the articles : we have noted more than one repetition, and have failed to find in their appropriate places many subjects referred to in other portions of the work. A still more serious defect is the sad lack of propor-

tion displayed in the space assigned to the various contributions—a manifest fault which detracts from the value of some of the best work. To cite a single example, without meaning to judge of the value of an article by its bulk, unilateral facial atrophy has more space assigned to it than the disorders of speech. Aside from these defects, the work is of great value and maintains a high average of excellence, many articles being of exceptional merit and hardly any absolutely poor.

Turning from the work as a whole to the individual contributions, of which we have space for but little more than a mention, the first two articles, which are introductory, are by Dr. E. C. Seguin—"General Semeiology of Diseases of the Nervous System; Data of Diagnosis," and "The Localization of Lesions in the Nervous System." To these articles nothing but praise can be given. Concise almost to a fault, they present in the limited space assigned to them a store of facts and a full account of the important symptoms in nervous diseases and the latest results of our knowledge of localization.

Dr. C. F. Folsom furnishes the next article—"Mental Diseases." In the too limited space at his command, he has wisely decided to touch lightly upon most of the forms of mental disease—except general paralysis—and to dwell mainly upon the relations of the general practitioner to insanity in its professional and social bearings, and the general principles of the treatment of the insane at asylums and at home. We regret that more space could not have been given to this subject, for, with a fuller discussion of the individual forms of insanity, this would be the best treatise on the subject for the general practitioner in English. As it is, we are glad to know that it has been printed separately.

Dr. C. K. Mills has four interesting papers on "Hysteria," "Hystero-epilepsy," "Catalepsy," and "Ecstasy." The first two form the most valuable contributions to the study of hysteria that have been made outside of la Salpêtrière. The author seems to us to make rather too sharp a distinction between the two affections, however, in stating that voluntary purposive attacks are to be classed as hysteria, and involuntary, non-purposive seizures as hystero-epilepsy. Full as these articles are of interesting details and valuable observations, we must regard them as unduly diffuse for the work before us.

Dr. H. C. Wood contributes a brief but valuable and suggestive paper on "Neurasthenia," and Dr. Henry M. Lyman an interesting but rather long paper on "Sleep and its Disorders." Dr. Wood also writes on "Acute Affections produced by Heat," giving the substance of his former well-known researches with considerable fresh matter. Dr. Wharton Sinkler gives a good account of "Headache," and Dr. S. Weir Mitchell contributes an admirable paper on "Vertigo." Dr. Sinkler also furnishes well-written articles on "Tremor," "Paralysis Agitans," "Chorea," and "Athetosis." Of these we can make special note only of the one on "Chorea," as



being a careful study of the affection, enriched with many valuable observations.

Dr. Allan McLane Hamilton's article on "Local Convulsive Disorders"—that is, Thomsen's disease, facial spasm, torticollis, and eclampsia—seems to us utterly inadequate and far below the standard of our present knowledge. He also furnishes the paper on "Epilepsy," which is much superior. It is an intelligent and carefully studied treatise, although we could have wished a fuller exposition of Hughlings-Jackson's theories, even at the expense of losing his scholarly review of the older ideas.

Dr. Morris J. Lewis, who writes on "The Neural Disorders of Writers and Artisans," gives us a very long paper, which is, nevertheless, one of the best in the volume, and the most exhaustive treatise on the subject of which we have any knowledge. He dwells especially upon the ætiology and symptoms of telegraphers' cramp, which has too often been regarded as of exceptional occurrence.

Dr. P. S. Conner furnishes an excellent paper on "Tetanus." This is followed by a paper, seven and a half pages long, by Dr. E. P. Davis, on "Disorders of Speech." We do not wish to criticise this paper too severely, for Dr. Davis writes intelligently on stuttering and stammering, and we do not know who is at fault; but when we recall the work done on aphasia since Kussmaul's great monograph appeared, and the fulness with which it is treated in ordinary text-books, such a neglect in a work of this sort, amounting almost to an absolute omission of aphasia, is inexcusable and disgraceful.

Dr. James C. Wilson's paper on "Alcoholism" merits the highest praise for its exhaustive classification of symptoms, its careful research, and its admirable good sense. His papers on "The Opium Habit and Kindred Affections" and "Chronic Lead Poisoning," are of equal value, but in the last-named article we are surprised to find no mention of lead as a cause of chronic myelitis or of the detection of lead in the urine as a means of diagnosis.

Dr. Mills gives a good account of "Progressive Unilateral Facial Atrophy;" Dr. Francis Minot furnishes four able and scholarly papers on "Diseases of the Membranes of the Brain and Spinal Cord," "Tubercular Meningitis," "Chronic Hydrocephalus," and "Congestion, Inflammation, and Hæmorrhage of the Membranes of the Spinal Cord;" and Dr. John Ashhurst, Jr., writes on "Spina Bifida."

Dr. E. C. Spitzka contributes a long paper on "Anæmia and Hyperæmia of the Brain and Spinal Cord." He gives an admirable discussion of the vexed question of vascular changes in the brain, and does much to put our knowledge on a firm foundation, but we would question whether this subject is of enough relative importance to be discussed at such length, to the exclusion of other matter. We cannot omit a word of praise for his admirable essay on acute myelitis. Dr. Spitzka also writes on "The Chronic Inflammatory and Degenerative Affections of the Spinal Cord" in a way that is unexcelled. Unfortunately lack of space demands too

great brevity in his account of some of the affections, but the section on *tabes dorsalis*, for example, is admirable for its exhaustive character, is careful research, and its deep learning.

Dr. Wm. Hunt gives a brief but interesting article on "Concussion of the Brain and Spinal Cord," in which he combats the former theories, without, however, discussing the subject in full.

Dr. R. T. Edes contributes one of the ablest articles in the work on "Intracranial Hæmorrhage and Occlusion of the Cerebral Vessels, Apoplexy, Softening of the Brain, Cerebral Paralysis," an article crowded with personal observations and research, and filled with the results of a wide range of reading.

Dr. H. D. Schmidt writes briefly of "Atrophy and Hypertrophy of the Brain;" and Dr. H. C. Wood on "Syphilitic Affection of the Nerve Centres," an article richly illustrated by classified tables of cases. He treats the subject under three heads, gummatous brain syphilis, syphilis of the cerebral cortex, and spinal syphilis.

We know of no articles more elaborate and exhaustive on the subjects of which they treat than the two papers by Dr. C. K. Mills and Dr. J. H. Lloyd on "Tumors of the Brain and its Envelopes," and "Tumors of the Spinal Cord and its Envelopes." The former is enriched with a table giving a synopsis of a hundred selected cases, the latter with a similar table of fifty cases.

Dr. Mary Putnam Jacobi contributes an elaborate article on "Infantile Spinal Paralysis," crowded with references to the literature of the subject, and filled with valuable matter which is dwelt upon with such detail as to be almost confusing.

Dr. H. D. Schmidt treats of "Disease of One Lateral Half of the Spinal Cord," and also contributes an acceptable paper on "Progressive Labio-glosso-laryngeal Paralysis."

Dr. F. T. Miles writes on "Diseases of the Peripheral Nerves" in rather an inadequate fashion. We have already spoken of the omission of an account of the individual forms of peripheral paralysis, but in addition to this, his remarks on the pathology of the peripheral nerves is unsatisfactory and incomplete, his discussion of the electrical reactions is obscure and misleading, and he omits to describe alcoholic neuritis.

Dr. J. J. Putnam contributes a full and systematic article on "Neuralgia," which is thoroughly satisfactory except for the necessary omission, from want of space, of the articular neuralgias.

The final paper, on "Vaso-Motor and Trophic Neuroses," is by Dr. M. Allen Starr, who contributes a valuable and scholarly account of the various symptoms and affections which are now classed under this heading, but which, as he says, will some day be put under other headings. His discussion of the vexed question of trophic neuroses in particular is an admirable contribution to our knowledge.

P. C. KNAPP.

**A Text-book of Medicine**, by DR. A. STRUEMPELL. Translated by H. F. VICKERY, A.B., M.D., and P. C. KNAPP, A.M., M.D., with Editorial Notes by F. C. SHATTUCK, A.M., M.D. New York : D. Appleton & Company, 1887.

Every book has its day. Niemeyer is on the wane, and Struempell in the ascendant. The former's book has attained a good old age, and laden with all the honors that a full generation of students could bestow upon it, it is now ready to leave the arena to others. Few text-books have wielded so potent an influence as Niemeyer's in its day; but in spite of its ten or a dozen editions, it is dying of old age, and all the tinkering of Seitz, himself a distinguished investigator, could not avail to keep it above ground. Struempell was right in his judgment that there was sore need of a text-book which would take into account the vast advances that had been made during the last decade in every branch of clinical medicine. The first edition of Struempell's work was published about four years ago, at a time when the medical world was full of Koch's discoveries, and our views on the causation of disease were destined to undergo great changes.

Few men could have acquitted themselves better of this task than Struempell has. With all the enthusiasm of a young investigator, he has managed to present the salient features of every disease in the strongest light; to be explicit without being verbose, and to exhibit originality in the treatment of a subject without entering upon the field of pure speculation.

While Struempell commands an excellent knowledge of the entire field of medicine, he is nowhere more at ease and more successful than in his description of diseases of the nervous system. And with this part of the book we are chiefly concerned. Of the nine hundred and fifty odd pages in this text-book, three hundred are devoted to nervous diseases—not at all a disproportionate number, if we remember that the author's original work lies entirely in this direction.

Like Gowers, the author begins with the simple and proceeds to the complex. First we have sections on the different forms of sensibility as an introduction to the chapter on diseases of the sensory nerves, with full descriptions of the various forms of neuralgia; then diseases of the motor nerves, prefaced again by general remarks upon disturbances of motility, and these followed by sections on the various forms of peripheral paralyses, of localized spasms, and a very full chapter on multiple neuritis. Vaso-motor and atrophic neuroses, diseases of the spinal cord, diseases of the medulla oblongata, diseases of the brain and its meninges, and neuroses without known anatomical basis (including epilepsy, chorea, athetosis, paralysis agitans, Thomsen's disease, catalepsy, hysteria and neurasthenia, and others), follow in the order named.

The distinguishing characteristics of these chapters are that they are written with the utmost clearness, that mere speculative theories are excluded as far as possible, that the clinical picture is in each instance developed with remarkable skill, and that special

stress is laid upon the pathology of the individual form of disease. According to our American notions, the remarks on treatment are rather meagre, but if they are limited, they are at least direct to the point, often suggest novel methods, and do not raise false hopes as to the curability of these diseases.

There is no need, we believe, to examine the many chapters in which controversial subjects are treated with a view to fixing the author's individual opinion in each instance; we have space for a few notes only, and it must be remembered, furthermore, that, in keeping with the purpose of the book, the author refrains, as a rule, from alluding to matters still *sub judice*.

Lead paralysis he holds to be a peripheral disease, though in some cases there may be an affection as well, of the spinal cord. Arsenical paralysis is in all likelihood peripheral. In all forms of neuritis, the toxic origin is the most frequent. Progressive muscular atrophy is treated of among spinal-cord diseases, and the author believes that there is a distinct spinal form and that the degeneration is confined to that portion of the motor-conducting tract which extends from the ganglion cells of the anterior cornua to the muscular fibres themselves. Pseudo-hypertrophy and simple juvenile atrophy he considers pure myopathies. Struempell does not believe in primary lateral sclerosis as a distinct disease, an anatomical entity; it is a convenient designation for a frequent group of symptoms.

Ophthalmoplegia progressiva is considered not unlike bulbar paralysis, and is discussed very briefly in the same chapter with the latter. As was to be expected from other publications of his own, S. compares the pathological process of poli-encephalitis to that of acute polio-myelitis in children, and makes "porencephaly" dependent upon a preceding poli-encephalitis. The author may be right in this, but at best it is rather a *previous* remark for a text-book.

As regards the etiology of locomotor ataxia, the author has introduced his most recent views of the formation of an intermediate *chemical* poison as the result of syphilitic infection. And so we might continue quoting the author's views. A little more caution in the expression of his own personal views would at times have been wiser; but the intelligent student will not be misled by these remarks, and the thinking practitioner will be benefitted by such suggestions.

That the book is a good one there can be no doubt, and that it is a thoroughly *useful* book the writer is glad to admit, for during the past two months he has made it his first book of reference in all matters neurological, and in every instance has found it satisfactory. And this is true not only of the neurological portion, but also of the chapters on pulmonary, cardiac, and renal diseases, which the present writer has consulted as occasion demanded.

Drs. Vickery and Knapp deserve more than a passing word of praise for the manner in which they have performed their part of the task. They have had the great advantage of translating a

work which was written about as clearly as German medical men are ever apt to write. But, granting this, it is to their credit that they have succeeded in making a readable and thoroughly reliable translation. It is only occasionally that they have fallen into slight inelegancies, and very rarely into actual error. On p. 682, we do not like the statement "that cerebral hemianæsthesia does cause hemiopia." The original reads, "Dass die Sehstörung bei der cerebralen Hemianæsthesie auch hemiopischer Natur sei." One clinical symptom (hemianæsthesia) cannot be the cause of another clinical symptom (hemiopia). "To say absolutely nothing," p. 597, is not very elegant. But these are very trivial errors, and count for naught in view of the evidence to be found on almost every page that the translation has been done in the most painstaking manner. The index is a model of its kind. Dr. Knapp, who is responsible for the neurological chapters, has added a few excellent notes to the various sections. We wish he would have given a little more "local coloring" at times to his work. Teplitz, Wiesbaden, and Rehme are recommended to complete the recovery from neuritis, myelitis, etc. The American student and practitioner have a right to know of some places nearer home that will answer the same purpose. Of Dr. Shattuck's share in this work we have nothing to say, as he appears to have steered clear of neurology.

The book is well printed, and not unwieldy. The illustrations are good and plentiful. We commend this text-book to all practitioners, and to students of neurology in particular. B. S.

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## Editorial Notes and Miscellany.

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We are pained to announce the death of Dr. James Stewart Jewell, of Chicago, on April 18th, after a lingering and complicated illness. During the past year or more, numerous evidences of Dr. Jewell's poor health reached his New York friends and acquaintances, but none expected so early an end to a bright career. Dr. Jewell was born September 8th, 1837, at Galena, Ill. He took his degree at the Chicago Medical College in 1860, and two years later began his practice in Chicago, in which he continued until the time of his death. From 1864 to 1869, he held a position as professor of anatomy in his college, and since 1872 has filled the chair of Nervous and Mental Diseases. In 1874, Dr. Jewell founded this JOURNAL, and, in the face of many discouraging conditions, continued it until a few years ago. Through the efforts of its founder, the JOURNAL soon obtained an enviable reputation among journals on neurology. Looking back upon past numbers,

we find that Dr. Jewell performed his editorial duties in the most painstaking fashion. Innumerable reviews, signed and unsigned, were written by him, and many excellent original articles appeared in the JOURNAL from his pen. Dr. Jewell had the satisfaction of seeing his JOURNAL a pronounced success, and although it had passed out of his hands, he retained a lively interest in its welfare up to his dying day. In 1886 Dr. Jewell founded another journal, the *Neurological Review*. The plan and design of the new journal were both good, but the editor's health was not equal to the task he undertook, and that journal had to be abandoned after three numbers had appeared.

Dr. Jewell rendered many valuable services to American neurology, and helped to place this special department upon an equal footing with other great specialties. He was one of the early members and organizers of the American Neurological Association. In practice Dr. Jewell was eminently successful, and deservedly popular among patients and physicians.

Dr. Jewell possessed great enthusiasm for his special subject, and was at all times well abreast of the latest advances in the science. Free from feelings of personal envy, he was ever happy to prove to others his recognition of the good work they were doing. We mourn the loss of an earnest student, a generous friend, and an honest critic.

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A great change appears to have come over our much-respected English contemporary, *Brain*, since it has assumed its official connection with the Neurological Society of London. The first number of the official journal contains but one original article, that by Bastian, on "The Muscular Sense; its Nature and Cortical Localization." This interesting and somewhat stirring article covers eighty-nine pages; upon this follows a discussion of fifty pages, in which Ferrier, Sully, Ross, Crichton-Browne, Hughlings-Jackson, Horsely, Haycraft, Mercier, and de Watteville participated.

The editor states that the character of the journal will remain essentially the same; but we do not believe that the editor will be able to give much space to contributors who are not members of that society. We should be sorry if *Brain* were to forfeit its international character. But whatever change may come over *Brain*, we account it a fortunate circumstance that English neurologists are now to meet face to face. The first discussion proves that Ferrier-worship is doomed, and that there will now be honest and fearless criticism all around. We intend at an early day giving a full *resumé* of Bastian's paper and of the discussion which followed the reading of the paper.

THE  
Journal  
OF  
Nervous and Mental Disease.

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Original Articles.

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SUBACUTE SPINAL PARALYSIS (PROGRESSIVE)—CASE WITH AUTOPSY.<sup>1</sup>

BY

C. L. DANA, M.D.

NEW YORK CITY.

THE history of subacute spinal paralysis will have to be rewritten, in the light of recent discoveries regarding multiple neuritis. At present, the cases heretofore regarded as of this kind may be divided into three classes.

I. Those of subacute multiple neuritis.

II. Those having a course like that of a subacute cornual myelitis, with a tendency to recover—subacute regressive spinal paralysis.

III. Those having a similar course at first to the preceding, but tending to progress, and develop finally a course like that of progressive muscular atrophy—*subacute progressive spinal paralysis*.

This classification, which is given by Gowers, will doubtless have to be changed in time. The following case illustrates the fact that a case, starting out clinically

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<sup>1</sup> Presented at a meeting of the N. Y. Neurological Society, December, 1886.

like a form of subacute cornual myelitis, progressed steadily until death.

For the case and specimen, I am indebted to my friend, Dr. Hermann M. Biggs.

Anton S., tailor, æt. 53; nat. Austria.

Admitted to Ward 18, Bellevue Hospital, Aug. 24th, 1886, in Dr. Biggs' service.

History of patient previous to present sickness negative. Patient had never been sick before; family history good. No history of syphilis.

On admission: Patient large, well nourished, and apparently had lost no flesh. Physical examination of chest and abdomen negative.

Pat. had lost almost entirely power over his lower limbs. He could raise them from the bed when lying on his back, but could not support himself on his legs and, if left standing unassisted, would fall immediately to the floor. There was also marked loss of power in the upper extremities, but not so great as in the lower. There did not seem to be any more inco-ordination in his movements than would result from the loss of power over the muscles. There was complete loss of tendon reflexes. There was a moderate degree of general anæsthesia in the lower extremities, with one or two spots of hyperæsthesia on the inner surface of either leg. The action of bladder and rectum was normal, and had been throughout his sickness.

Patient said that he first noticed a weakness in his legs four or five months before his admission; said that this gradually grew worse until about one month before he entered the hospital, when he was obliged to give up work and remain most of the time in bed. Said he had had no pain at any time, and complained of nothing excepting the loss of power in his limbs. There was no affection of any of the special nerves or of cranial nerves. The batteries at the hospital were all out of order, and no electrical reactions could be tested.

Examination of urine negative.

Patient was placed on large doses of iodide of potas-



sium. He, however, gradually lost more and more power over his extremities, but otherwise remained in same general condition until about two days before his death, when he developed a slight fever, became delirious, failed rapidly, and died in partial coma on Sept. 21st, not quite one month after his admission.

At the *autopsy*, nothing of importance was found in any of the thoracic or abdominal viscera. Brain showed nothing. There was a deep injection of the small vessels of the pia mater. There were no lesions in the cord or its membranes visible to the naked eye, except a small cavity about one-twelfth by one-sixteenth inch in diameter in the right half of the intermediate gray and right cornu. The cavity extended from about the sixth cervical to the third or fourth dorsal nerve.

*Microscopical examination.*—The cord was hardened in Müller's fluid in an incubator. After ten days it was placed in alcohol. Sections were made in the middle and lower cervical region, upper and middle dorsal, and the middle lumbar and sacral segments.

The lower cervical and upper dorsal sections showed the upper limit, the middle part, and the lower limit of the cavity described above.

*Middle lumbar region* (3d to 4th).—The white matter shows a considerable number of small colloid bodies, especially in the peripheral part of the lateral and posterior columns. These bodies indicate a shrinking or total loss of the nerve fibre.

Large spaces in the posterior columns indicate excessively dilated blood-vessels which have been torn away. In the lateral columns are numerous vessels with decidedly thickened walls. One may count with a two-third-inch objective twelve distinctly-marked distended vessels in the left lateral column, ten in the right. There are eight large spaces made by vessels torn away in the posterior column.

In sections of healthy cords, I have observed about three-fourths this amount of vascularity.

*Gray Matter.*—The antero-lateral group of cells is

shrunk, and has nearly disappeared on one side. The central group is also affected.

The other anterior cornual groups are not markedly changed. One may observe several large multipolar cells in the central part of the posterior gray cornua.

The gray matter is richly studded with distended capillaries and small vessels. The vascularization in the posterior horns is striking. The vessel walls are not notably thickened. The central canal shows nothing peculiar.

The *middle dorsal region* shows the same changes as above. The colloid bodies referred to, however, are still more numerous. The vascularization of the gray matter is very marked, and there are very few healthy cells in

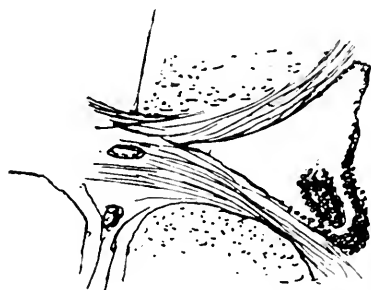


FIG. 1.—A cavity in central gray of lower cervical region, showing blood-vessel and cell infiltration.  $\times 400$ .

the anterior horns. The cells of Clark's column are shrunk or gone, and there is much vascularity in this region and in the intermediate gray anteriorly to it. The fine nerve mesh-work brought out by Weigert's stain in lower sections is very indistinct here.

Sections in the lower dorsal region show Clark's columns to be normal.

Sections in the sacral segments show nothing abnormal.

Sections in the middle cervical region show a capillary injection of the intermediate and anterior cornual gray matter. At one point in the intermediate gray, there is an open space whose walls are surrounded by layers of small round cells. Sections lower down reveal this as de-

veloped into a well-marked, triangular-shaped cavity lined with cells (see illustration).

Sections in the upper dorsal region show the cavity well developed in the anterior horn of the right side. The right half of the cord is smaller and flattened antero-posteriorly (see cut).

The white matter of the lateral columns is richly studded

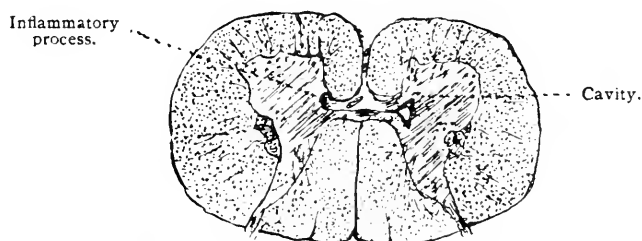


FIG. 2.—Middle cervical region, showing upper limits of lesion.  $\times 3$ .

with the small colloid-like bodies. There is no marked vascularity. The nerve-cells in the left anterior horn are present only in the antero-internal group. In the region of Clark's column there are a few cells which do not look normal, and there is much vascularity at these places.

The left anterior horn is nearly gone, as shown in the cut. The wall is clean and smooth. At certain levels in

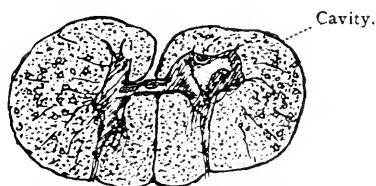


FIG. 3.—Upper dorsal region, showing extent of cavity.  $\times 3$ .

some sections, however, a cellular infiltration could be seen.

A few anterior cornual cells are left. The posterior horns show a great degree of vascularity, with thick-walled vessels and cellular infiltration around them.

A study of the sections shows that the process began in a congestion, dilatation of vessels, pouring out of cells,

breaking down of surrounding tissue, finally obliteration of vessels and loss of nerve substance. In other words, a slow inflammatory process, beginning in the central gray matter, forming a central focal myelitis, which was gradually extending upwards and downwards. If the patient had lived, other focal points would have doubtless developed, or the process would have extended downward continuously.

The case is one, then, of central focal subacute myelitis, with the formation of cavity.

It is not, strictly speaking, peri-ependymal, for that process is not centred about the central canal.

The case is interesting in connection with the pathology of subacute spinal paralysis.

It also bears upon the question of cavity-building in the cord and of syringomyelia. If anything, it tends to support the views of Hallopeau, Schüppel, Schuele, Eickholt, and others who contend that syringomyelia may be the result of a myelitis.

This above subject has been so thoroughly gone over recently by Oppenheim (*Charité Annalen*, 1886, p. 408) that it need not be taken up here.

## A THIRD CONTRIBUTION TO THE STUDY OF LOCALIZED CEREBRAL LESIONS.<sup>1</sup>

BY E. C. SEGUIN, M.D.

I OFFER the two subjoined cases as additional evidence of a localization of motor functions in limited areas, cortical and subcortical, of the cerebrum. The first case more especially demonstrates the results of a superficial irritating lesion of the cortex.

While separately considered, these cases may not be of very great value, yet, taken together with the evidence already accumulated, they seem worthy of record.

CASE I.—Relating to the face-centre. In collaboration with Dr. J. L. Hicks.

Summary: Right hemiparesis, most marked in the right cheek; clonic convulsions in right cheek; slight fever; no aphasia or choked disk. Recovery after prolonged use of iodide of potassium. Sixteen months later, death after symptoms of acute meningitis. Autopsy shows a patch of simple adhesive meningitis over a part of left motor area, and recent acute tubercular meningitis.

A boy aged 7 years, named Harold, very intelligent and healthy, seen at Flushing in consultation with Dr. J. L. Hicks, Jan. 13th, 1885.

For about two weeks before this date, it was noticed that the child was not as well as usual—even before Christmas his right hand felt numb at times, and an examination of his copy-books revealed a change in his handwriting; less steady writing, an inequality between different words, in contrast to previous remarkable uniformity of the script. An attack of measles appeared Dec. 23d (1884), and ran a very mild course. During this week of illness, the patient several times complained of his right hand "going asleep," and feeling "queer."

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<sup>1</sup> Read at the second annual meeting of the Association of American Physicians, June 3d, 1887. For first and second contributions, *vide* Seguin, "Opera Minora," p. 202 and p. 495. New York, 1884.

On Jan. 2d, after slight fever on the preceding day, it was observed that he spoke thickly, that saliva dribbled from the right buccal angle, and that the right cheek and arm were decidedly paretic; the leg slightly so (?). There was neither headache nor aphasia; the mouth temperature ranged from  $98.5^{\circ}$  to  $100^{\circ}$ . After cutting the gums over some molar teeth, improvement appeared.

On Jan. 9th, one hour after a very mild faradic application to the weak muscles on the right side, there occurred clonic convulsive movements in the right cheek, with suspension of speech and turning of eyeballs to the right. No spasm in other parts; patient fully conscious. This *facial monospasm* lasted for more than two hours. No return of convulsive movements since; the temperature has ranged from  $98.5^{\circ}$  to  $99.5^{\circ}$  (in mouth); the right face (upper lip near median line especially) and the right hand have occasionally felt numb and "queer." Speech has been thick at times, and a few times he has used the wrong word. No gastric disturbance, but constipation has been present.

Inquiry into possible causes reveals no serious injury to the head, although a "slight" (?) fall on knees, arm, and forehead from a velocipede occurred about three months ago. No pulmonary, renal, or aural disease.

*Examination.*—Mind clear, speech normal (it was thick a little while ago); no headache or cranial tenderness to percussion. Only lesion in eyes is great myopia, with posterior staphyloma and large veins. Right cheek is paretic, the tongue deviates to the right; the right hand is weak, and in walking there is a slight drooping of body to right. The knee-jerk is low on both sides, but more marked on the right. No anesthesia. Heart normal. Mouth temperature  $100^{\circ}$ ; pulse 90, regular.

The *diagnosis* was uncertain as between a tumor in the left pre-central gyrus (its lower third and adjacent part of second frontal gyrus) or a localized meningitis over the same part. The idea that such very localized ("Jacksonian") symptoms could be reflex from irritation about the gums I could not entertain.

*Treatment.*—Fluid extract of ergot in doses of 5 drops, and saturated solution of iodide of potassium, 10 drops every three hours. Absolute rest and plenty of liquid food. The iodide to be rapidly increased.

Jan. 27th. The iodide has been given in doses of 75 drops (about 5 grams) three times a day. Great improvement has occurred; occasional numbness of right cheek and hand. Iodide to be reduced.

Feb. 16th. Paresis of right cheek is constant, though the arm is stronger and well used, and no spasm has occurred. No disturbance from the iodide, now used in doses of 30 drops *ter die*.

April 29th. Except paresis of right cheek, has been remarkably well until yesterday, when the following occurred: a numbness began in right forefinger, extended into the arm, to the right side of face, right half of tongue, and right heel (not toes!). There

was no spasm. The right upper lip was decidedly swollen, and there was drewling from right corner of mouth. No aphasia, headache, or fever. To-day, the cheek is paretic, but not more than before the attack, and there is still some prickling in the right hand. Otherwise well; advise rapid increase of iodide to 45 drops *ter die*.

May 28th. Except right facial paresis, has been well until 18th inst., when an attack, like the one above described, occurred (similar except that there was no numbness in the leg). There was another attack on the 19th. Excitable. On 22d, had attack of severe transverse frontal headache of short duration. Is taking 65 drops of iodide *ter die*; and this was subsequently increased to 100 drops *ter die* (about 20 grams per day) without unpleasant effects.

Examined again June 13th. Distinct right hemiparesis, most

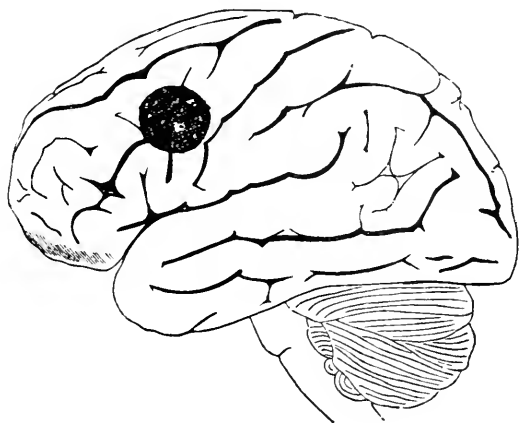


FIG. 1.—Patch of localized adhesive meningitis over the second frontal and precentral gyri of the left hemisphere in case 1.

marked in the face; no anæsthesia; speech and optic nerves normal. General condition good, except pallor. Advised stop medicine; patient to be gradually allowed more liberty and watched carefully.

Oct. 3d. Has been very well. On June 17th, a slight attack of numbness occurred. The paresis has passed away, except that, when tired, the right cheek droops somewhat. It is to be observed that, although numbness appeared first in the hand in some of the attacks, the cheek was the seat of the greatest and most persistent paresis, and that the clonic convulsions occurred only in the facial muscles (the lower set); so that it might reasonably be concluded that the lesion of the left hemisphere was chiefly in or upon the cortical face-centre.

The next year, early in April, 1886, the lad developed unmis-

takable symptoms of acute general meningitis: headache, photophobia, vomiting, slow and irregular pulse, temperature of  $101^{\circ}$  and  $102^{\circ}$ . May 1st, delirium set in with higher temperature, and death occurred the next day. In this attack, there were no convulsive or paralytic phenomena. The ophthalmoscope was not used.

The autopsy, on May 4th, revealed the ordinary recent lesions of tubercular meningitis. This was intense over the pre-pontic base of the brain, over the apices of the temporal lobes, and well up into both fissures of Sylvius. There was much gelatinous effusion about the chiasm. The rest of the brain showed no evident meningitis. The ventricles were largely dilated and filled with clear fluid. The cerebral substance, especially its cortex, was pale. Microscopic examination showed abundant cellular infiltration of the pia, and many globular and muff-like tubercular masses along the blood-vessels.

The most interesting result of the autopsy was finding the lesion which had caused the symptoms of the previous illness. This was a patch of intimate adhesion, without evident exudation, between the dura and the pia (or its arachnoid layer), situated over the ventral part of the left precentral gyrus and the adjacent (caudal) end of the second frontal gyrus, over a space one inch (25 mm.) in diameter. Although quite firm, the adhesion gave way to traction without decortication. It was a *localized adhesive meningitis* situated over the face-centre, and impinging upon the arm- or hand-centre. It was wholly dorsad of the speech-centre, and decidedly ventrad of that part of the precentral gyrus in which lesions cause primary and predominant symptoms in the fingers and hand.<sup>1</sup>

While the face-centre was the seat (in the first illness) of the chief irritation and greatest malnutrition (anæmia), it is evident from the symptoms that there was more or less extension of irritation (very much as in electrical experiments in animals) to the rest of the motor area of the same side.

It seems to me that, taken together with other recorded cases, this case goes to support the view that in the human brain the cortical centre for the face is in the caudal end of the second frontal gyrus, where it is continuous with the precentral gyrus.

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<sup>1</sup> Compare Case IV. of first contribution for intermittent aphasia caused by localized meningitis over Broca's speech-centre; and Case I. of second contribution for brachial monospasm and monoplegia caused by a tumor in the middle part (vertically) of the precentral gyrus. "Opera Minora," p. 211 and p. 495.



CASE II.<sup>1</sup>—Bearing on the question of the location of the leg-centre.

Summary: Paresis of right leg; clonic spasm in right abdominal muscles; right hemi-epileptic attacks with first spasm in abdominal muscles; then in the leg, last in the arm (none in face). Repeated similar attacks without loss of consciousness; paralysis of the right leg, paresis of right arm; no aphasia or choked disk. Death; sarcomatous tumor in white substance of left hemisphere, subcortical, situated dorso-laterad of the paracentral lobule.

Mr. F. W., aged 49 years; seen Nov. 22d, 1881. Has enjoyed good health, with exception of occasional headaches, which have been less troublesome in the last few years. Denies syphilis and injury to head.

About a year ago had severe left occipital neuralgia; the region midway between morbid process and median line being hyperæsthetic. Pain distinctly worse at night. This lasted one month and has not recurred. Was generally weak and in poor health during the past summer; legs felt heavy, and feet were "sensitive" in the morning. The right foot was weaker and œdematous; there was no numbness; and he was able to keep at work during the autumn.

Some four or five weeks ago, there was marked increase in the difficulty in walking. The right foot was dragged rather stiffly with the heel raised. Was unsteady. About Nov. 1st, after application of an electrical current, he had a first attack of spasm limited to the right abdominal muscles. A day or two later, there was a more severe seizure, spasm beginning in the right abdominal muscles, extending to the leg, and lastly to the arm of same side. No spasm in face, and no loss of consciousness. The right arm and leg were weak for half an hour after attack. Since, he has had numerous similar attacks, always on the right side, and without loss of consciousness; from six to eight seizures a day. Marked paresis of the right limbs. No aphasia, headache, vomiting, or mental disturbance.

Yesterday (Nov. 21st), in an attack, spasm extended to the right face and the left limbs; he lost consciousness (attack witnessed by Dr. Bruce, a relative).

*Examination.*—Pallor; heart and pulse normal; no aphasia. The right pupil is a little wider than left, both active; optic nerves normal.<sup>2</sup> There is no facial or lingual deviation, but the right arm and leg are very paretic. In the upper extremity, the paralysis is most marked in the region of shoulder and in upper arm, and he still has a fairly good grasp. When standing, the right foot is held in equino-varus position; and the knee-jerk is raised on this side. No anæsthesia.

<sup>1</sup> I am indebted to Dr. R. W. Amidon for many of the notes in this case, and for ophthalmological examinations.

<sup>2</sup> This was the case throughout the illness, an ophthalmoscopic examination being made shortly before death.

*Diagnosis.*—Probably a cerebral tumor. It is learned that the grandfather and one aunt of patient (both on paternal side) had "cancer."

*Treatment.*—Ord. thirty grains (2 grams) of bromide of potassium at bed-time. Iodide of potassium in large doses three times a day. Absolute rest.

Nov. 27th. More hemiplegia of a peculiar type. The leg is completely paralyzed, the upper arm and shoulder more paretic than the forearm and hand (grasp  $12^{\circ}$  on a stiff dynamometer); right cheek a little flabby. At night, has "cramps" in right leg.

Owing probably to the influence of the bromide of potassium, the convulsions did not recur, but there were frequent attacks of

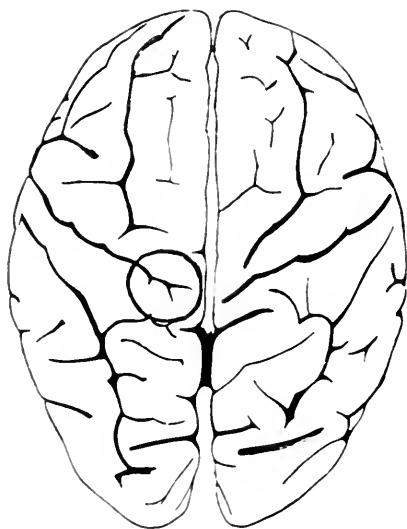


FIG. 2.

FIG. 2.—Diagram of convexity of brain—showing location of the (sub-cortical) tumor.



FIG. 3.

FIG. 3.—Diagram of transection of left hemisphere, showing the exact position of the tumor, and of two minute secondary growths.

painful cramp in the right leg at night (extension of foot and flexion of leg on thigh). The next day paralysis extended to the hand, but the tongue protruded straight; speech slow; no aphasia.

Dec. 4th. More voluntary power in hand and fingers. Marked aphasia and alexia (?); attacks of a syncopal nature occasionally. In one of these the patient died (Dec. 6th), with a low temperature<sup>1</sup> and stertorous breathing.

<sup>1</sup> No figures are given in the original notes, but I am very sure that the temperature was not subnormal; there was simply absence of the hyperpyrexia which usually prevails in the last stage of cerebral tumor.

*Autopsy.*—Dura normal; arachnoid shows some opacities, more over the left hemisphere. The convolutions of the left parietal lobe are flattened; the flattening not extending further frontad than one-half of the frontal lobe, and ventrad not reaching the inferior parietal lobule. The occipital lobe is slightly flattened. The whole hemisphere appears swollen, and larger than the right. Transections reveal a sarcomatous globular tumor ventrad of the top of the pre- and post-central gyri, and dorso-laterad of the paracentral lobule. The growth is mainly in the white substance, but invades the cortex, though it does not appear externally. Two very small nodules of the same new-formation are found further ventrad in the white substance. No other lesions in the brain.

The lesion interrupted fasciculi of fibres connected with the paracentral lobule and with the upper (mesal) ends of the pre- and post-central gyri.

It is very interesting to note the relative immunity of the forearm and hand from symptoms.

From this case we obtain, I think, additional proof that the innervating centre for the leg is in the paracentral lobule, and perhaps also in the mesal ends of the pre- and post-central gyri, in accordance with what has already been established.<sup>1</sup>

Another conclusion, of more novel interest, which may be drawn from this case, is that the cortical centre of innervation for the muscles of the shoulder and upper arm is probably in the upper part of the pre- and post-central gyri, between the forearm and leg centres.

We may also infer that the centre for the abdominal muscles is in the same area, but nearer to the leg centre.<sup>2</sup>

I desire to add, by way of record, summaries of two cases, already published.

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<sup>1</sup> Compare Case II. of the second contribution, "Opera Minora," p. 499.

<sup>2</sup> I have at present under observation, in consultation with Dr. O'Gorman, Jr., of Newark, a most interesting case analogous to the above. The attacks of "Jacksonian" spasm are on the right side and chiefly affect the muscles of the shoulder, upper arm, and side of chest (probably only in the serratus and pectorals, but not in the intercostals). In a few attacks the spasm has extended to the whole of the right side, and one or two general convulsions with loss of consciousness have occurred. No choked disk is present, and there is but little headache. The case is improving under mercury and iodide of potassium, with some bromide of sodium.

## CASE III.—Relating to the visual half-centre.

Male, aged 46 years ; seen in 1884. Malignant endocarditis ; sudden attack of left lateral hemianopsia, with (temporary) left hemi-numbness ; no anesthesia or paralysis. Death in May, 1885, from progress of endocarditis ; multiple embolisms, pseudo-intermittent fever, etc. Autopsy showed softening of the right cuneus and fifth temporal gyrus, caused by embolism of the occipital artery (and other visceral lesions). This diagnosis of seat and nature of lesion had been made during the patient's life.

Published in full in this JOURNAL for January, 1886, p. 25.

## CASE IV.—Relating also to the visual half-centre.

Male, aged 42 years ; seen in October, 1885. Left lateral hemianopsia ; slight staggering in gait ; temporary paresis of both external recti ; choked disks. Absence of headache, paralysis, and anæsthesia. Operation by Dr. R. F. Weir, March 9th, 1887 ; removal of an enormous sarcomatous tumor situated on the inner aspect of the right occipital lobe, probably connected with the ventral part of the falx cerebri and thus injuring the cuneus early in its development. The diagnosis of tumor of the right cuneus was made by me in November, 1885, sixteen months before the operation.

This case subsequently passed under the care of Dr. W. R. Birdsall, by whom and Dr. Weir it has been very ably reported in the (Philadelphia) *Medical News*, April 16th, 1887.

## Clinical Cases.

### TUBERCULAR MENINGITIS—DISSEMINATED MILIARY TUBERCULOSIS.

WARDS FOR NERVOUS DISEASES OF THE PHILADELPHIA HOSPITAL. SERVICE OF CHARLES K. MILLS, M.D.

M. B., colored, aged 7 years, a thin, ill-nourished child, presenting a well-marked angular curvature in the mid-dorsal region of the spine, on the afternoon of September 2d, while begging, fell in the street in convulsions. She was picked up and brought in the ambulance to the Philadelphia Hospital, and on admission there, a few hours after the convulsion, she was perfectly conscious, answering questions freely and complaining only of pain in her head. During night of the same day convulsions recurred, continuing with short intervals of repose until near midnight, when, after a short inhalation of amyl nitrite they ceased entirely, the patient passing into a deep sleep. The convulsive movements consisted of irregular tossings of the arms and legs, clenching of the hands, and rolling of the eyes, these movements being accompanied by irregular and stertorous or semi-stertorous respiration.

On the following day, September 3d, the child was stupid, lying with half-closed eyes and knit brows; she was inclined to be irritable, and complained of headache when aroused; her bowels were constipated; appetite was lost; temperature was normal. She was ordered sodium bromide with syrup of the iodide of iron. She became brighter, and for several days showed marked signs of improvement.

On the afternoon of September 8th, convulsions again occurred; the temperature rose to  $101^{\circ}$  F.; she had continuous pain in the head; she was also troubled with vomiting, which was controlled with difficulty; the bowels were obstinately constipated. She was ordered a fever mixture with small doses of quinine, and her bowels were opened with an enema.

During a period of ten days, but little change occurred in her condition; she still complained of headache, vomiting occurred at irregular intervals; her temperature varied from normal in the morning to  $100^{\circ}$  F. in the evening. The following is the temperature record from the 8th to the 18th of September inclusive.

September	8th.—Morning,	$100^{\circ}$	F.;	Evening,	$101^{\circ}$	F.
"	9th.—	"	$100^{\circ}$	F.;	"	$100.4^{\circ}$ F.
"	10th.—	"	$99^{\circ}$	F.;	"	$99.4^{\circ}$ F.
"	11th.—	"	$98.2^{\circ}$	F.;	"	$98^{\circ}$ F.

September 12th.	—Morning,	100° F.;	Evening,	99.4° F.
"	13th.	— " 95.4° F.;	"	98.3° F.
"	14th.	— " 98° F.;	"	98° F.
"	15th.	— " 99° F.;	"	100° F.
"	16th.	— " 98.8° F.;	"	99.5° F.
"	17th.	— " 99.4° F.;	"	100° F.
"	18th.	— " 99° F.;	"	98.5° F.

On September 19th, her temperature ascended in the evening to 102°. Severe convulsions occurred during the night. The following day she lay in a stupor, although she could be aroused. Her face wore an anxious, pinched expression. Respirations were rapid and shallow; the pulse was rapid and feeble. Belladonna and bromides were administered. She continued in much the same condition, but gradually failing. September 23d, convulsions again occurred during the night. On the 24th, she had difficulty in swallowing, with increased stupor. On the 25th and 26th, the stupor deepened, and she had irregular movements of the arms and legs, with labored respiration. On the afternoon of the 27th, her temperature steadily ascended, reaching 106° half an hour before death, which occurred at 8.15 P.M. The temperature record from the 19th until her death was as follows :

September 19th.	—Morning,	99.2° F.;	Evening,	102° F.
"	20th.	— " 96.2° F.;	"	102° F.
"	21st.	— " 102.4° F.;	"	100.2° F.
"	22d.	— " 100.4° F.;	"	99.4° F.
"	23d.	— " 99.8° F.;	"	100° F.
"	24th.	— " 100.4° F.;	"	99° F.
"	25th.	— " 98.3° F.;	"	98.1° F.
"	26th.	— " 101.2° F.;	"	100.3° F.
"	27th.	— " 99.3° F.;	4.45 P.M.,	106° F.

*Autopsy.*—The autopsy was held twenty-two hours after death. There was turgescence of the meningeal vessels. The cerebro-spinal fluid was largely increased. Numerous aggregated, semi-translucent miliary tubercles were found in the pia mater and arachnoid, more particularly in the occipital and parietal regions along the course of the vessels. Here and there were small, irregular patches. The longitudinal fissure anterior to the corpus callosum was obliterated. A few indistinct tubercles were to be seen along the course of the vessels in the Sylvian fissure. Anterior to the optic chiasm was a collection of sub-arachnoidal lymph. The anterior and posterior horns of the left lateral ventricle were slightly enlarged, and contained about two drachms of fluid. Both pleural cavities were obliterated by extensive and firm adhesions. The left lung showed very numerous disseminated tubercles. The heart showed nothing abnormal, except several sub-endocardial ecchymoses on the ventricular wall of the left side. In the abdomen were evidences of widespread tubercular peritonitis. The liver and spleen contained numerous miliary tubercles; the kidneys showed a small number of miliary tubercles in the cortex. Numerous isolated glands were turgescient and semi-translucent.

## REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILA- DELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF W. H. WALLACE, M.D., PHYSICIAN  
IN-CHIEF, AND CHARLES K. MILLS, M.D., CONSULTING PHYSICIAN.

### CASE XIV.—Puerperal Mania.

Reported by Harriet Brooke, M.D., Assistant Physician.

L. W., aged 29 years, married, negress, house-servant, was admitted to the hospital August 31st, 1886. When admitted, she was a tall and strong-looking colored woman, neatly dressed. Her expression and manner were noisy and excited. Her pupils were equal, normal in size, and responded to the light. She had no symptoms of general or ordinary paralysis. Physical examination of the thorax or abdomen was impossible on account of the patient's excessive activity. She was constantly jumping, dancing, and singing. Her pulse was rapid, and her tongue slightly coated and pale. Examination of the urine showed it to be normal in color, reaction acid, with no albumin. The patient had given birth to a male child two weeks before her admission to the hospital. The labor was not abnormal, but very rapid, the delivery taking place before the arrival of the physician. She had no convulsions.

This was her first attack of insanity. A maternal aunt had had an attack of puerperal mania which lasted two years. No other history of insanity in the family was obtainable. The patient had been married twice. She had one child by her first husband, a girl about eight years old. She had been married to her second husband about a year.

About four or five days before she was brought to the hospital, her husband said she commenced to act strangely. She showed a disposition to wander from home, was sleepless at nights, wild and excited in manner at times, and abusive to her baby. Finally, she left her home in Delaware, and took a train to Philadelphia. While in the cars she was excited and noisy, and when she arrived at the Broad street station she was taken in charge by the police after making quite a disturbance.

The following are some extracts from the record book of the hospital:

September 6th, 1886.—Her mental condition was one of excitement. She still danced and sang, denuded herself completely, was

incoherent in speech, and sleepless. Hyoscine hydrobromate, gr.  $\frac{1}{4}$ , was given her at bed-time.

Sept. 13th.—She has much improved since her admission; talked rationally about most things, and was almost abnormally bright and quick. In many of her remarks she was still much exhilarated.

Oct. 2d.—She complained of pain in the hypogastrium, the urine was scanty, and she had pain on micturition. She was ordered ext. buchu fl., spts. juniperis co., and spts. ætheris nitrosi three times daily.

Oct. 21st.—She was slightly quieter than last week. Potas. brom. and chloral were given at bed-time.

March 9th, 1887.—She has much improved mentally, quieter and less excitable, had lost some of her unsystematized delusions, and was quite industrious and helpful about the ward. She had not attempted to escape this last month.

April 20th.—She had entirely recovered, and had been paroled. She talked freely of what she now recognized were her delusions.

Although it is not a common occurrence for patients to escape from the hospital, this patient made several escapes which were remarkably well planned and executed in spite of every precaution. October 15th, 1886, the record says that she escaped again in spite of ordinary precautions, and was returned to the hospital. She was always more excited after an escape, and since the last time she had been secluded or restrained much of the time, if there had not been a nurse who, after finishing her work in the ward, could devote her whole attention to her.

November 12th, 1886.—The following notes were entered: A few nights ago the patient succeeded in getting the night nurse in her room, stealing the keys, and locking her in, after which she climbed up on a wardrobe, and from there to a transom, crawled through it, and dropped down outside, and would have got away, but she was observed and brought back by the night watchman.

The next night she succeeded in lighting a rag by one of the gas jets, and setting fire to some straw beds that were piled together at one end of the ward ready for use. This she contrived to do in the two or three minutes that the nurse was preparing her room for the night, and had it not been for the courage and quickness of the nurse, who succeeded in smothering the flames with a large double blanket, the house would probably have been burned. When questioned as to her motive for firing the place, she said that in the commotion created by the fire she wanted to make her escape; later, she denied having anything to do with the fire.

It is worthy of note that after an attack of mania lasting nearly seven months, the convalescence, when it commenced, was a very short one; and also that there was marked exaltation of the intellect as well as of the emotions—the patient composing impromptu and singing jingling verses, many of them remarkably bright and witty, a feat that she was totally unequal to in her normal condition.



## CASE OF ABNORMAL CHOREA WITH COPROLALIA.<sup>1</sup>

By E. C. SEGUIN, M.D.

This is, I believe, the second case of this rare disease reported in this country. The first was presented to this Society by our President, Dr. C. L. Dana, a little over a year ago, under the title of "Convulsive Tic with Explosive Disturbances of Speech."<sup>2</sup> Echolalia, present in many of the cases of Charcot and his pupils, was wanting in this case.

Geo. F. K., aged 13, seen at the Manhattan Eye and Ear Hospital February 25th, 1887. Was a healthy baby; no convulsions. Parents healthy. Is one of ten children; one died at birth, another (a boy) died of "water on the brain" at age of 18 months. A sister (aged 36 years) has been epileptic from 8th year.

Five years ago, developed general common chorea. This was soon aggravated, and for weeks the patient had paroxysms of running and shouting. No better particulars can be had. Ever since he has had more or less jerky chorea, at times better, at others worse. Last autumn, began to ejaculate obscene words or short phrases, with jerks of limbs. This "coprolalia" has continued until a few days ago.

The movements (abnormal chorea or convulsive tic) consist in sudden electric jerks of parts, such as shoulders, arms, or neck (bowing movements), occasionally snuffles or snorts. Some movements are apparently in abdominal muscles and diaphragm, though inspiratory spasm is very rare. The legs seldom jerk. The movements are present on both sides, occurring sometimes on one side, sometimes on both sides. Facial muscles occasionally jerk. Each movement is almost lightning-like, and very suggestive of a cortical discharge.

The ejaculations have been equally sudden, loud, and perfectly uncontrollable. They issue simultaneously with a muscular jerk. Bad or obscene single words, or short sentences of two or three words are uttered. As examples, we may cite: "Mother's dead," "Theresa has a fit" (referring to epileptic sister who may not be having a fit), "damn it," "sh—t," etc. In the last three months, dating from a few days before first examination, he has said no formed words, but

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<sup>1</sup> Read at the New York Neurological Society meeting of June 7th, 1887.

<sup>2</sup> Vide this JOURNAL, 1886, p. 407.

only a loud "hein" or "hah" sound, uttered mostly in the throat, mouth rarely open. Patient has been greatly mortified by these utterances, and ardently desires relief.

Examination. Choreic movements, with grunting sound as above. Pale, fairly well-grown boy. Both cranium and palate well-formed. No cardiac lesion. Is very intelligent and speaks well. No paresis or anæsthesia. Patellar jerk very feeble on both sides.

The treatment has consisted so far in the use of solution of chloro-phosphide of arsenic, gtt. iij., gradually increased to gtt. xij. *ter die*. Valerian given freely without effect. Since March 14th, has taken from 2 to 4 tablets of  $\frac{1}{100}$  grain of crystallized hyoscyamia a day with very decided relief. Almost quiet for hours at a time. In the last two weeks no medicine. The boy is worse to-night (more jerking and louder throat sound) than I have ever seen him.

I would suggest that if we are to regard the common muscular movements in this case as due to cortical discharges from the motor centres for the various muscular groups affected, it is not unreasonable to suppose that the ejaculations of formed words represent discharges from Broca's speech centre in the caudal end of the left third frontal gyrus, while the inarticulate laryngeal and pharyngeal sounds are due to discharges from the centre for laryngeal movements in the caudal end of the *right* third frontal gyrus.

## Periscope.

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PHYSIOLOGY (INCLUDING PHYSIOLOGICAL PSYCHOLOGY.

### **The Muscular Sense.** (*Revue Philosophique*, April, 1887.)

At the February session of the *Société de Psychologie Physiologique*, MM. E. Gley and L. Mariller presented some facts bearing on the much-vexed question of the muscular sense, and the feeling of effort. The experiments were made on a patient who had completely lost sensibility in the upper part of his body as far down as his umbilicus. He perceived neither contact, nor heat or cold, nor pressure, nor pinching, nor twisting of the arms; and electrical stimulation was absolutely without effect. The sensibility in the lower part of the body was preserved although obtuse. The experiments were as follows: 1st. His eyes having been bandaged, his arms were placed in many different positions without his knowing that their position had been changed. The arm was flexed and extended alternately without his perceiving it. The patient having placed his hand on his knee, the hand was taken away without his knowing it and raised above his head, the hand of the experimenter being placed on the subject's knee; the patient thought his own hand was on his knee. Unable to measure the amount of power that he uses, he breaks the objects that he handles when he does not look at his hands. 2d. His eyes being bandaged, a weight of 2 kilogram. was attached to his wrist, the forearm being flexed horizontally, and his elbow being placed on the edge of the table, in the manner of Donders and Van Mausveldt for studying the muscular elasticity. The string being cut the weight fell (without noise), and the arm quickly flew back, but the patient was not conscious of any movement and thought his arm had not moved. Similar experiments made with weights of 100 gm., 200 gm., 500 gm., 1, 2, and 5 kilogram. gave the same result; he perceived neither the movement of the arm, nor the effort necessary to hold up the weight, nor the difference in the weights.

3d. Asked to lift three similar closed vessels (two empty and weighing 250 gm., the third filled with mercury and weighing 1,850 gm.), he says all three are of equal weight. Repetition of this on succeeding days gives the same result. His eyes being bandaged, he is asked to lift a weight of 11 kilogram., but he does not perceive that he is holding a very heavy object and lets it go without attempting to retain it in his hand.

4th. With his eyes closed he cannot distinguish between a piece of modeller's wax, a piece of very hard wood, a large rubber tubing, and a folded and wrinkled newspaper, feeling no difference in resistance and not perceiving that he held anything in his hand.

This experiment appears very important to the authors as showing that the disappearance of the sensibility of the skin and of all the subjacent parts carries away also the feeling of resistance, that is to say, that form of muscular sense that has especially served the classical psychology to uphold its theory.

5th. His forearms were tied very tightly to a table so that he could not move them. He was then asked to fold his arms and to say when he had done this. He always thought that he had completely succeeded in folding them, whereas he moved them only slightly; his reason for thinking that he had accomplished the movement was because of the time that he had occupied. This last experiment is not cited as a proof of the non-existence of the muscular sense, since alone it is susceptible of two interpretations; but in view of the preceding experiments this interpretation is thought to be the only legitimate one. It is important to note that the statements of the patient himself show the importance of the notion of time in the appreciation of movements, since the indications furnished ordinarily by the sensations are lacking. These experiments are held by the authors, and with good grounds, to show that the disappearance of the superficial and deep sensibility carries with it the disappearance of the muscular sense, since, if he has still, with his eyes closed, some appreciation of movements, it is due especially to the knowledge of the time it takes to effect them, and perhaps also to an obscure consciousness of the modifications of respiration. If some movements may still be accomplished—and they can only be performed imperfectly when the sight does not direct them (*motor memory*)—this is due on the one hand to habit and on the other to the motor power of the images. The experiments add to the weight of cumulative evidence against the theory that the feeling of effort is due to a feeling of innervation ("*innervationsgefühl*"), and go to show that it is due to *afferent* sensations "coming from the tense muscles, the strained ligaments, squeezed joints, fixed chest, closed glottis, contracted brow, clinched jaws, etc." (Prof. James).

WILLIAM NOYES.

### **The Muscular Sense; its Nature and Cortical Localization.** By H. C. BASTIAN, M.D. (*Brain*, April, 1887).

The object of this long and exhaustive article, recently read before the Neurological Society of London, is to prove that the so-called motor centres of the cerebral cortex are in reality the cortical termini of muscular-sense impressions.

Starting from the proposition that all purposive movements are guided by sensations or by afferent impressions of some kind, the author proceeds to discuss these "kinæsthetic impressions." Impressions of various kinds combine for the perfection of a sense

of movement, viz., cutaneous impressions, impressions from muscles, and other deep textures of the limbs (such as fasciæ, tendons, and articular surfaces), all of which yield conscious impressions of various degrees of definiteness; whilst, in addition, there seems to be a highly important set of unfelt, or but little felt, impressions which guide the volitional activity of the brain, and which seem to bring it into relation with the different degrees of contraction of all the muscles that may be called into action. By means of these impressions we are made acquainted with the position and movements of our limbs, we are enabled to discriminate between different degrees of resistance and weight, and by means of them the brain derives much unconscious guidance in the performance of movements generally.

Kinæsthetic impressions are not instigators of movement in the same sense that visual or auditory impressions may be, but they are the guides of movement, and their guiding influence is brought to bear, partly under the form of actual sensations and partly under the form of revived impressions (the memories of past activity). It is these kinæsthetic impressions which awaken in consciousness the muscular sense.

The muscular sense may be impaired or lost by disease, and Bastian cites a number of cases of various kinds in which this loss has been a prominent symptom. In these cases, the common feature is an inability on the part of the patient to judge of the locality and position of his limb when his eyes are closed, or to execute voluntary movements with accuracy when not aided by sight. It occurs in locomotor ataxia and in some cases of cerebral hemianæsthesia. But Bastian calls attention to the well-known fact that the degree of disturbance of muscular sense in these diseases is independent of the degree of disturbance of tactile and painful sensations, and urges that this independence proves that these sensations are independent of one another and that the tracts by which they are transmitted are also distinct. He claims that we are ignorant of the course of these tracts, while those who are familiar with German and American literature will recollect that Kahler, in Prague, and Spitzka, in New York, as long ago as 1884 established independently the existence and situation of the muscular-sense tracts—a fact which was confirmed by a collection of cases published in this JOURNAL, in July, 1884, by the reviewer. But Bastian has also found a number of cases in which cortical disease has been attended by a loss of muscular sense, and since such disease corresponds in its course with diseases of the motor area, and since the lesions in these cases have been found in the motor area, he claims that the kinæsthetic impressions are received in this part of the brain. He then shows that such impressions may be revived in memory; in other words, that we possess memories of motion as well as of sensation—a fact which will be admitted at once by those who are familiar with Meynert's writings upon this subject, in which he constantly uses the term "*bewegungsvorstellungen*"—motor memories. In fact, much of this part of Bas-

tian's article coincides in a remarkable degree with the positions long ago advanced by Meynert, although no reference is made to the German author. Such a revival of a kinæsthetic impression he considers identical with the sense of effort. It is here that he comes in conflict with a different view. Wundt believes that "the strength of the sensation of effort is dependent only on the strength of the motive influence passing outward from the centre which sets on the innervation of the motor nerves." Bain holds that "the sensibility accompanying muscular movement coincides with the outgoing stream of motor energy, and does not, as in the case of pure sensation, result from an influence passing inwards by ingoing or sensory nerves;" and to this view Hughlings-Jackson adheres.

Bastian, however, believes that "the feeling of expended energy, by which we obtain our ideas of resistance and of an external world, is not contained, as we think, in the volitional act itself, but is derived from impressions emanating from the moving organs themselves during the actual accomplishment of movements." He cites Prof. Wm. James, of Harvard, in his support. In reply to the question as to the nature of our sensible perceptions of movement, James says: "I unhesitatingly answer, an aggregate of afferent feelings coming primarily from the contraction of muscles, the stretching of tendons, ligaments, and skin, and the rubbing and pressing of joints; and secondarily, from the eye, the ear, the skin, nose, or palate, any or all of which may be indirectly affected by the movement as it takes place in another part of the body. The only idea of a movement which we can possess is composed of images of these, its afferent effects. The degree of strength of our muscular contractions is completely revealed to us by afferent feelings coming from the muscles themselves and their insertions, from the vicinity of the joints, and from the general fixation of the larynx, chest, face, and body, in the phenomena of effort, objectively considered. When a certain degree of energy of contraction rather than another is thought of by us, this complex aggregate of afferent feelings, forming the material of our thought, renders absolutely precise and distinctive our mental image of the exact strength of movement to be made and the exact amount of resistance to be overcome."

All our knowledge of movement, therefore, comes to us through sensory centres, and motor ideas are not in any way connected with an efferent or outgoing current. It is these kinæsthetic centres which are located in the so-called motor area of the brain. The muscular sense presides over voluntary movements, but the co-ordination of the motion is wholly a matter of spinal-cord action. The movement depends on the spinal cord or subcortical centres, though its particular *qualities* of force, rapidity, duration, etc., are dependent upon the cerebral or volitional influence. The cerebrum may be said to exercise a kind of co-ordination—it co-ordinates or adapts the movements which are organically represented in the spinal cord so as to make them accord qualitatively with the aim conceived. But to do this, it must be instructed from

moment to moment as to the exact nature of the movement actually produced, and this it gets from sight, touch, and the muscular sense. When movements are complex, long practice is needed to acquire the kinæsthetic impressions necessary, as in learning to play on an instrument.

Thus the performance of a voluntary act is always preceded by an idea or conception of the movement we desire to execute; and this idea or conception is for ordinary movements compounded of two kinds of past impressions, namely, those of the visual sense and those of the kinæsthetic sense. It is an error to look for special motor centres for the production of voluntary movements of any kind, either in the cortex or elsewhere. Ferrier's so-called "motor centres" are in reality kinæsthetic centres in which "muscular-sense" impressions in particular have been registered. As to the objection that from these centres the pyramidal tract proceeds which degenerates downward, Bastian replies that the pyramidal tract is doubtless efferent; but this fact does not at all touch the question whether the ganglion-cells which exercise a trophic influence on such efferent fibres constitute parts of sensory or of motor centres. The sensory incitations to movement constituting part of the volitional act must pass off from certain cortical areas in a definite and orderly manner, in order to excite motor centres, wherever they may be situated, and these pass along the pyramidal tract.

It may seem of little importance whether the Rolandic area is called a "motor area" or not, especially as Bastian admits that from it issue volitional motor incitations. But this he denies, for he says we should not call a cortical centre for afferent impressions motor any more than we should call the cell-nuclei on the sensory side of a spinal reflex arc "motor;" and again, the retention of such nomenclature tends to foster such false notions as that in the sense of movement we have to do with a so-called active sense differing from other modes of sensibility, and that we have such things as "motor ideas."

For these reasons, it seems necessary to Bastian to admit that the excitable areas in the Rolandic and marginal regions of the cortex are in no proper sense of the term "motor centres," and that the evidence at present in our possession makes it extremely probable that they are termini for kinæsthetic impressions derived from muscles, so that their excitation in this or that region is the immediate precursor of this or that kind of voluntary movement.

The article is one which does not admit of condensation and should be carefully read by those interested in the subject. The discussion of it before the Society was participated in by Drs. Ferrier, Sully, Ross, Crichton-Brown, Horsley, Haycraft, Mercier, and De Watteville, and elicited a number of varying views as to the relations of the muscular sense and of effort to volitional movement and its mechanism.

M. A. STARR.

**The Nature of Muscular Activity.** DR. VON KRIES (*Arch. f. Anat. u. Phys.*, Supplement, 1886); HORSLEY & SCHAEFER (*Journ. of Phys.*, 1886).

The activity of the neuro-muscular system manifests itself in two ways, sensation and motion. These may be said to be the fundamental categories of neurology, and to them corresponds the division of the spinal cord into posterior and anterior halves. When we shall discover how the activity of the afferent nerve passes into that of the efferent nerve, the nervous system will have yielded up its most hidden secret. In the mean time, the physiologist must be satisfied with tracing as far as he can the nature of each of these activities separately. The problems of sensation have been largely given over to the psychologist, while physiological methods are best applicable to the study of motion.

The action of our nervous system, by which it causes a muscle to contract, is usually called innervation. The question that at once suggests itself is whether this innervation power has any limits. How rapidly can our will send down messages to the muscle and have these messages obeyed? Dr. von Kries has recently answered this question by recording the quickest possible movement which the finger can make. The finger was moved down and up as in striking a piano key; and as the object was to send out an impulse to bring the finger back as soon as possible after the down stroke was innervated, the minimum interval between two innervations was measured. First, let us speak of the time of the motion including a down and an up stroke. This was found to be from  $\frac{1}{4}$  to  $\frac{1}{11}$  of a second. If, instead of moving the finger voluntarily, it is caused to move by electric irritation of the muscle, the time is longer. This fact, that we can move voluntarily faster than when our muscle is contracted for us, is very important for the theory of muscle action. The movement does not of course stop abruptly when the finger has returned to its initial position; but only this portion of the movement is taken into account. The quickest such movement of the middle finger is .077 sec., of the hand .074 sec. It is rather curious that the extent of the movement has little effect on the time; the movements of medium length tend to take least time. Now the portion of this time from the beginning of the down stroke to the beginning of the up stroke, *i. e.*, the minimum innervation interval, is .061 sec., while the up stroke consumes only .017 of a second. The innervation interval is never shorter than  $\frac{2}{9}$  of a second. In trilling on the piano, we have a good example of this sort of movement; and the fastest trill which ordinary music requires is not more than 13 to a second. Experts can keep up a short trill at nearly 20 per second. As regards movements of other pairs of antagonistic muscles, the time of the tongue movement is  $\frac{1}{15}$  sec., of the foot and the jaw  $\frac{1}{8}$  to  $\frac{1}{6}$  sec.

If, however, such a movement is to be performed rhythmically for many times its speed must be diminished slightly. The finger can move in this way ten times per second (after practice), the jaw only 6.2, and the foot seven times per second.



If, instead of recording the resulting motion of the finger, we record the swellings of the contracting muscle, we gain a deeper insight into the nature of the contraction. We have, in the first place, from such a record, that the activity of the muscles continues after the motion is over and slowly falls back into its quiescent state; and secondly, the innervation and rhythms leave their trace in the curve of contraction. Normal muscular contraction is generally considered to be tetanic, that is, it is not a simple muscular twitch, but a series of maximal contractions. The curve that the swelling of the muscle unites is not a simple up stroke followed by a down stroke, but is composed of several smaller curves of this nature: it shows a rhythm, and a quite constant one, of about ten to twelve per second. It is to be understood that when we move the finger once, however slowly, the muscle gives a series of rapid twitches; what we think is a single innervation is really a manifold series. And in the shortest possible movement there are about four such waves in the curve of contraction, each such wave consumes about one-fifteenth of a second, the curve, of course, continuing after the resultant motion has gone into effect.

Finally, in rhythmical movements, the smaller waves of contraction become variable within wide limits to about forty per second.

These results are confirmed and supplemented by the recent experiments of Horsley and Schäfer. They excited electrically the cortex, the underlying motor fibres, and the motor area of the spinal cord of dogs and other animals, and found the same result from each of these modes of stimulation, viz., a contraction rhythm of about ten per second in the muscle. They varied the frequency of the stimulus from ten to fifty per second, but found that the rhythm of contraction was (generally) independent of the frequency of the stimulation. They found, too, that the same holds for voluntary and reflex motions as for these artificially excited ones.

The most natural explanation of these facts is that the innervations are surmounted in the motor cells of the cord (probably), and are re-issued from those cells at a constant rate of ten per second, no matter at what rate these cells receive these impressions.

Dr. Schäfer has applied the same method of study to voluntary contractions in man with the same result.

We see, then, that there is an innervation rhythm in the nervous system itself, which limits the rate of our quickest motions, and which determines, independently of our will, the activity of our muscles. What to our consciousness is a simple motor act, is really a series of contractions rhythmically exploding themselves at the rate of about ten per second.

J. JASTROW.

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#### PATHOLOGY OF NERVOUS SYSTEM.

**"Paralysis of the Isthmus of Panama. Beriberi, Kakke."** PIERRE MARIE. (*Progrès Médical*, p. 168, 1887.)

The author, in this article, after briefly reviewing the paralytic

symptoms occurring in beriberi, as described by Scheube in his monograph, goes on to describe a case of paralysis of the lower extremities occurring after a sojourn of several weeks on the Isthmus of Panama, which in the opinion of the writer is certainly a case of beriberi. Cases of this kind have been seen by Charcot, and one also by Hutinel. Marie's case is that of a male, *æt.* 37. Family history unimportant. Patient was in good health until the occurrence of this affection. On the 29th of August he arrived at Colon, where he remained during his entire stay on the Isthmus, with the exception of several visits to Fox River and Monkey Hill (places in which fever is very prevalent). Between the 20th and 25th of September he noticed that he was getting weak, had that his health was growing worse from day to day, although he was unable to assign the cause of his ill-health to any particular organ. The physician who saw him at that time said he was suffering from severe *anæmia*. During the last few days of his stay at Colon he was exceedingly weak, and fainted several times. During this time his abdomen was very much distended, and seems to have been the seat of an effusion of fluid. The patient also observed the existence of *anæsthesia* over the abdomen near the umbilical region, the *anæsthetic* part being of the size of about two hands. On the 11th of November he set sail for home (France). The first day on the ocean he noticed an abundant purpuric eruption on both feet, from tibio-tarsal articulations to the middle of the calves. The second or third day of the trip the legs became *œdematous*. Until this time no pain, and no special weakness of the legs. On the 22d of November, on attempting to rise, he noticed that both legs were paralyzed. Neither at this time nor subsequently was there any affection of the bladder. Seven or eight days after the occurrence of the paralysis in the legs, the patient noticed a well-marked weakness in both thumbs; the other fingers were not affected. This weakness lasted about two weeks and was not accompanied by any disorder of sensation. The paralysis of the legs began to improve about the 25th of December.

Examination of the patient showed atrophy and flaccidity of the muscles of the thighs. Paresis of certain groups of muscles of the lower extremities, and weakness of the abductors of the thumbs.

Reflexes were all normal, with the exception of the patellar tendon reflex, which was entirely absent. Mechanical excitability of the muscles seems decreased.

Sensation is almost normal, except at the lower part of the legs, where it is slightly diminished. No vaso-motor symptoms. Pupillary reflexes normal.

This case seems to differ from typical cases of beriberi in the rapid development of the paralysis, as well as in the short exposure (about ten weeks) necessary to its production; but both of these abnormalities have been exceptionally noticed by other observers.

The most interesting question is that of the geographical dis-

tribution of this affection. It is apparently new that beriberi is endemic at the Isthmus of Panama, and expressions of opinion by physicians living there would prove very valuable.

G. W. J.

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**Paralytic Ataxia of the Heart of Bulbar Origin.** M. SEMMOLA. (*Gazette des Hôpitaux*, p. 881, 1886.)

In 1876, Semmola published his first observations upon the influence of the nervous system in the production of affections of the heart. In 1881, at the International Medical Congress of London, he clearly formulated his views upon the existence of a cardiac affection, slowly produced as a result of faulty innervation of the oblongata and of the cardiac ganglia. The present communication is the result of further observations upon this subject. All the patients observed were free from any rheumatic, gouty, or syphilitic taint. The results of S.'s observations may be summarized as follows: The prodromal period generally occurs between the forty-fifth and sixtieth years, that is to say, at a time when, in addition to the general impressionability of the nervous system, two other classes of functional disorders are apt to occur: those of the heart and those of the stomach. This period may last for two to three years, and is characterized by a weakness of the systole with an increase in the number of heart beats. This demonstrates simply an insufficient power of recuperation on the part of the nervous system, an exhaustion of the pneumogastric and of the ganglia which preside over the functions of the heart. During this prodromal period the affection is susceptible of a complete cure, if absolute rest and absence of exciting causes are procured. If, on the contrary, the causes persist, the affection enters upon the confirmed stage, curable in some cases, which is characterized by the following symptoms:

- a. Palpitations. Dysystole. Respiratory difficulties.
- b. Development of a mottled coloration of the hands, arms, and legs. There is no œdema. This is a purely neuro-paralytic stasis.
- c. Respiratory uneasiness developed after the slightest exertion. Auscultation reveals the existence of crepitant râles at the base of the lungs; these are also due to a neuro-paralytic stasis.
- d. Attacks of suffocation which awaken the patient and oblige him to sit up, when they soon disappear. These differ from the attacks of suffocation occurring in organic affections of the heart, by the absence of true dyspnœa, by their short duration, and by the impossibility of detecting any change in the heart itself.

Semmola believes that this respiratory difficulty is caused by the anæmia of sleep, during which condition the bulbar centres of respiration become still more exhausted until paralytic effects ensue.

- e. Development of œdema in the feet. The dysystole becomes permanent, and all the grave symptoms of organic heart disease appear.

G. W. J.

**Trophic Alteration and Spontaneous Shedding of the Nails in an Hysterical Woman.** I. FALCONE. (Quoted from *La Riforma Med.*, August, 1886, in *Rivista Clinica*, October, 1886.)

After the observations of Joffroy and Pitres regarding the spontaneous shedding of the nails in ataxia, as well as the numerous examples of individuals so afflicted when suffering from general paralysis, disseminate sclerosis from gout, etc., the writer feels justified in adding this case of similar trouble in an hysterical subject.

A woman, 50 years old, of good heredity and excellent constitution, had always been healthy until 1870, when she lost her son. Her character changed entirely. She became irascible, sad, and commenced to complain of left hemiparesis, then of dyspepsia, colic pains, recurrent moroseness, then transitory paralysis presented itself in the extremities, hyperæsthesia, and wandering paræsthesia, then coughing with sense of constriction, aphasia, globus hystericus, salivation, and a profuse sweating of the lower extremities. All these symptoms persisted in turn from 1870 to 1880, and did not disappear after a long journey, which had been prescribed, except the cough.

In November, 1885, the patient visited the tomb of her son, which event made a great impression upon her, and resulted in the return of the laryngeal symptoms of the hysteria, and with them psychical excitement.

A second trip dissipated the greater part of these symptoms. However, Feb. 16th, 1886, the husband observed that the patient's nails presented a curious appearance of fine rugæ and a loss of the healthy lustre, the most on the right thumb. Feb. 19th, the lady complained of slight formication in the right thumb and both of the great toes, and there was a formation of pus under these nails, and they were shed. After this there was a suppuration with abundant and fœtid pus, and in the space of two months the matrix and bed of the nails became covered with a new epidermis. The other nails remained in place. In them was observed a most active growth and a hardening; they desquamated easily, and appeared duller than in health.

In the process of repair, the nails of the toes grew from the matrix and recovered the appearance and consistency of health, while the nail of the right thumb was a proliferation of the bed of the nail *without any participation of the matrix*. It grew irregularly, with prominences of various sizes which crossed it in all points, and by the character of the form which it assumed seemed rather a drying up of the bed of the nail. GRACE PECKHAM.

#### THERAPEUTICS OF NERVOUS SYSTEM.

**A Case of Traumatic Epilepsy Cured by Trepanning**  
(*Centralb. f. Nervenheilkunde, Psych.*, etc., November 15th, 1886).

Dr. G. Völckers, house physician of the Luis Hospital, Aachen, reports the following case from the service of Brandeis:

Patient was a strongly-built man of 26, in robust health. In January, 1882, he was struck by a heavy limb of a falling tree. He felt but little pain, and started to walk home, but soon fell unconscious. He was confined to bed for three weeks, having been unconscious most of the time. On getting up he spoke confusedly, and acted foolishly. After five weeks there were no morbid symptoms present, and no evidences of a central disturbance could be detected.

In January, 1884, after two years of complete health, he suddenly lost consciousness while plowing. He remained unconscious ten minutes, with clasped thumbs and spasmodically drawn limbs. The attack was without any premonitions, and during the same day he had six others. He had a second attack in February, and a third in April, both being without aura.

Examination at the hospital showed an extended median depression of the skull, forming an oval that embraced the posterior part of the frontal bone, with the longest diameter seven centimetres in the direction of the sagittal suture; the transverse diameter was fully five centimetres.

There was no unevenness or roughness on the surface of the skull, and no abnormality in any organ of the body.

The temperature was normal; the pulse full and of normal frequency.

It was assumed that the attacks were caused by a diminution of brain-space caused in part by the depression, and in part by intracranial hemorrhage. Concussion of the brain was excluded because, in spite of the severity of the blow, the brain symptoms were first noticed a quarter of an hour after the injury, and also because of the long duration of the trouble.

After five weeks, all the symptoms had disappeared, the extravasation having been absorbed, and the brain having become accustomed little by little to the pressure of the depression.

A certain degree of compression may thus exist for a long time without interfering with the brain functions and without endangering life; but even a small intercurrent increase of intracranial pressure may produce grave symptoms of pressure. The severe work performed by the patient was a sufficient cause in this case.

An incision was made in the scalp following the direction of the depressed margin of the bone. Around the periphery of the depressed section ten holes were drilled, each one-half centimetre in diameter, and the intervening bridges of bone were broken down by a mallet and chisel, and the whole of the plate of bone lifted up. This bony plate was united with the dura, which appeared normal otherwise; because of these adhesions there was no hemorrhage from the dura.

After some hours, there was a slight vertigo and twitchings in the arm, but these disappeared after a few minutes. The headache present before the operation disappeared in two days, and

did not return. He was discharged twenty-four days after the operation. Two months after discharge he had an attack of vertigo while at work, but did not lose consciousness. He had no other attack. He was last seen two and one-half years after the operation, when he appeared strong and healthy, and considered himself entirely cured.

WILLIAM NOYES.

### **Treatment of Trigeminal Neuralgia by Pulverization of Methyl** (*Gazette des Hôpitaux*, p. 870, 1886).

"In trigeminal neuralgia, the first indication is the immediate alleviation of pain." "No agent seems more appropriate for this purpose than the chloride of methyl in refrigerant pulverization." The above propositions are advanced and discussed by Dr. Peyronnet de Lafonvielle. The time which has passed since the therapeutic application of this remedy by Debove in cases of sciatica is not long, but the number of cases so treated is now quite large. In August, 1885, Abadie published cases of cure of trigeminal neuralgia by this means. The observations of de Lafonvielle were made at Abadie's clinic, and it appears that even in the most severe cases these pulverizations produced almost immediate relief from pain. Frequently this occurred after the first application, but invariably after several. De L. has seen both acute and chronic cases cured by this means. Even when the pain was dependent upon some general or central cause, the relief was prompt and of long duration. The methods of applying this remedy seem to present certain difficulties. The low temperature which the chloride of methyl assumes when in gaseous form, and the strong pressure necessary in order to keep it in a liquid state at ordinary temperature, seem to constitute the chief obstacles. In the earliest applications, glass siphons, analogous to the ordinary seltzer-water siphons, were employed. But several explosions resulting, these glass siphons were replaced by receptacles of metal specially arranged for the purpose intended. The necessity of a special apparatus, the high cost of chloride of methyl, as well as the great care necessary in its application, are all factors which will render the introduction of this method into general practice almost impossible. Nevertheless, in view of the unsuccess of most other methods of treatment, these obstacles should not be considered too great.

G. W. J.

### **Dosage in Electro-Therapeutics.** R. VIGOUROUX (*Progrès Médical*, p. 29, 1887).

The author, after reviewing the former inadequate means of measuring the strength of the galvanic current by giving the number of cells used, goes on to explain the present method (absolute measurement), and claims that this also is insufficient, particularly in regard to the physiological effects of the current. His line of argument is as follows:

The resistance of the human body varies very much. In one

patient, it may be necessary to employ thirty-six cells in order to obtain a current of say 5 ma.; in another, twelve cells will produce the same result. Therefore, the two patients, although each receiving the same amount of electricity, have not been "electrified in one and the same manner." In the one, the "electrical pressure" or electro-motor force has been three times as great as in the other. Two currents having the same intensity may, from a physiological point of view, be entirely different. The chemical and magnetic effects of a current depend solely upon the amount of electricity in the circuit; the thermal and mechanical effects depend upon the electro-motor force with which the current is produced. Consequently, it is entirely faulty to consider only the first of these propositions and to neglect the second.

We must, then, always specify both the electro-motor force and the intensity of the current. It is, therefore, proposed by V. to describe the current used by employing two numbers joined by the word "for," the first number representing the number of volts and the second the number of ampères. If, for instance, the electro-motor force is twenty-five volts and the intensity fifteen ten-thousandths of an ampère (the author uses a ten-thousandth of an ampère, instead of a thousandth as a standard), this current would be described by saying that a current of "twenty-five for fifteen ten-thousandths" was used. In order to easily obtain these two factors, V. has modified a vertical galvanometer so that by means of a commutator the same instrument may be employed as a voltmeter, it having also a scale graduated in volts. (The above ideas have been noticed because they form the subject of an editorial, and great importance seems to be attached to them.)

The author entirely disregards the proposition of Müller to use Erb's electrodes with their number of square centimetres distinctly marked upon them, and then to express the current by a fraction, the numerator of which represents the number of milliamperes employed, and the denominator the number of square centimetres contained in the electrodes. This is much simpler than Vigouroux's proposition, and manifestly meets all requirements, for, knowing the density and the intensity of a current, we gain nothing by knowing the electro-motor force.)

G. W. J.

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### Mogiphonie.

B. Fränkel (*Deutsche Medic. Wochen.*, 1887, No. 16) describes, under the title mogiphonie, an affection of the voice in public speakers and singers which he likens to writer's cramp; and considers a functional neurosis. He gives an account of six cases of this affection, in all of which the ordinary act of talking was not affected, but as soon as attempts at singing, preaching, or loud speaking were made, the voice absolutely failed. An examination with the laryngoscope showed that the glottis, whose movements were normal in breathing and in ordinary speaking, was held open,

or rather failed to close properly when any attempts at producing a loud sound was made. Hysterical and neurasthenic conditions were not present, and the author is therefore forced to believe in the existence of an occupation neurosis of the larynx, similar in origin and symptoms to the other occupation neuroses, as writers', pianists', etc., cramp. In one case massage of the larynx was followed by recovery.

M. A. S.

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### Neuritis Puerperalis.

P. J. Möbius (*Münchener Medic. Wochenschr.*, No. 9, 1887) describes a number of cases of a peculiar kind to which he gives the name puerperal neuritis. As diphtheria and syphilis, and as lead and other poisons are capable of producing neuritis, whose chief symptom is a motor paralysis in certain groups of muscles, so according to Möbius a certain form of neuritis is liable to develop after childbirth and as a sequel to puerperal diseases. Its symptoms are both sensory and motor. Its location is in the ulnar or median nerves of the arms or sometimes in both together. The disease begins gradually with shooting pains in the hands, which increase to constant pain and paræsthesiæ. Occasionally spasms of the muscles ensue and frequently atrophy of the thenar eminence, and reaction of degeneration in the muscles supplied by the median and ulnar nerves develops. More rarely the muscles of the upper arm, as well as of the forearm and hand, are invaded. The duration of the disease varies greatly, but the prognosis is usually favorable, recovery ensuing sooner or later in the majority of cases. Inasmuch as paralysis following childbirth has usually been ascribed to a lesion of the spinal cord, the seven cases of Möbius, fully detailed in this article, are of much interest. It is not stated, however, by the author whether this form of neuritis is always preceded by some form of puerperal septicæmia or whether it may occur without any other symptoms of blood-poisoning.

M. A. S.

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### The Psychical Symptoms in Alcoholic Paralysis and Multiple Neuritis.

Korsakow (*Neurol. Centralbl.*, 1887, No. 9) records 20 cases of alcoholic paralysis dependent upon multiple neuritis in which marked psychical disturbance was noticed. He distinguishes three forms of mental aberration quite distinct from one another. In the first form, the mental change was chiefly characterized by irritable weakness. The patients were uneasy, irritable, excitable, discontented with everything and every one, and were thrown into fits of crying or of rage by the slightest opposition to their whims; they were sleepless and very restless.

In the second form, a true confusion of ideas began simultaneously with the sensory and motor disturbances. The patients did not recognize their surroundings or attendants, and were subject to



hallucinations and illusions—especially of sight—and even occasionally to delusions of persecution.

In the third form, there developed a very great disturbance of memory, especially of occurrences during the illness of the patient. There was little disturbance of consciousness and no pathological affection of the emotions in this form. During its progress, however, a gradual diminution of mental power supervened almost to the degree of imbecility, though throughout the weakness of memory was the most noticeable feature. With the gradual subsidence of the motor and sensory symptoms the mental powers returned.

In the second part of his paper the author describes four cases of multiple neuritis not of alcoholic origin in which psychical as well as nervous symptoms occurred. In three of these the disease followed some affection of the pelvic organs in females; in one after a miscarriage, in a second after puerperal parametritis, and in the third after a febrile disease of the pelvic viscera. The author is inclined to explain these on the theory of septic infection, and considers the connection of the neuritis and psychical symptoms as by no means accidental, since it has been noted by other authors. He terms these forms of psychical disturbance "neuritic psychoses." He believes that a given poison or infection affects both the brain and the peripheral nervous system, and he supports this theory by citing the similar well-known effects produced by lead, arsenic, and bisulphide of carbon.

M. A. S.

### Hereditary Progressive Atrophy.

Dr. J. W. Bennett, of Brookhaven, Miss., has recently described a remarkable example illustrating the family form of the disease. A summary of his paper was presented to the New York Neurological Society by Dr. C. L. Dana on June 7th. Photographs of cases were also shown. The accompanying table exhibits the succession of sufferers in this family.

#### DR. BENNETT'S FAMILY MUSCULAR ATROPHY.

GREAT-GRANDPARENTS healthy. One great-grand-uncle born with one arm.

GRAND-UNCLE. (Case V. Lived to old age. Childless.)	GRANDFATHER. (Case VI. Died æt. 78.)	GRAND-AUNT. (Case VII. Lived to old age. Childless.)
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MOTHER, æt. 44. (Case III. 13 children.)	UNCLE, æt. 42. (Case IV. 7 children, all well at present time.)
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GRANDSON, æt. 25. (Case I.)	GRANDSON, æt. 12. (Case II.)
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The atrophy showed itself in all these cases at puberty or shortly before; except in the case of the grandfather, whose dis-

ease began when he was twenty-two years of age. The atrophy in all cases affected muscles of the upper arm and shoulder, extending to the thoracic muscles. In the grandfather's case, late in life, the lumbar muscles became affected. In the cases of the grand-uncle and grand-aunt, it is stated that several years before death the hands and feet became "withered and cramped." The full text of the paper was read before the Mississippi State Medical Association, in April, 1881.

For tables of other families, comp. *Archives of Medicine* (N. Y.), IV., p. 319. A detailed account of another family is given by Hammond, "Treatise on the Diseases of the Nervous System," 7th ed., N. Y., 1881, p. 541. E. C. S.

## Review.

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**Elements of Physiological Psychology.** A Treatise of the Activities and Nature of the Mind from the Physical and Experimental Point of View. By GEORGE T. LADD, Professor of Philosophy in Yale University. New York: Charles Scribner's Sons, 1887, pp. 696.

Prof. Ladd's work is a most welcome addition to psychological literature. It is the first attempt in English to cover the ground of experimental psychology, and, appearing seven years after the second and last edition of Wundt's "*Grundzüge der Physiologischen Psychologie*," it supplements that epoch-making book, and brings clearly before us the advances that have been made since that time. It is a surprise as well as a pleasure to receive a book conceived in such a truly broad and scientific spirit from a professor of philosophy who is at the same time a doctor of divinity in a confessedly conservative religious body. Prof. Ladd has no fear of the study of the mind from the experimental and physiological point of view, and while he holds that there is no ground for extravagant claims, yet there is still less ground for any fear of consequences, and we may certainly agree with him that with declamation, either in attack or defense of the "old psychology" of the "introspective method," etc., one may dispense without serious loss.

The problem, then, that physiological psychology sets for itself is, "What is the nature of mind considered in the light of its correlations with the body? and, Do the so-called physiological and the so-called psychical phenomena belong to one subject or to more than one?" It is, of course, but the old, old question that each will solve for himself according to his environment and the light that he gets from the little lantern with which he sees his own small portion of the universe; but it is indeed refreshing to have it confessed that "introspective psychology, important as its results have been, and indispensable as its method is, has shown its incompetency to deal with many of the most interesting inquiries which it has itself raised. On the other hand, psychology as pursued by the experimental and physiological method has already thrown a flood of fresh light upon many of these inquiries."

As the field covered is an entirely new one for English readers,

it is fitting to indicate the ground that Prof. Ladd covers and the conclusions that he reaches.

Part I. would make a fair treatise on the physiology of the nervous system, and under the headings of "The Elements of the Nervous System," "The Combination of the Nervous Elements into a System," "The Nerves as Conductors," "Automatic and Reflex Functions of the Central Organs," "End Organs of the Nervous System," and lastly, "The Mechanical Theory of the Nervous System," the well-known facts of nerve anatomy and physiology have been set forth in a manner that is, on the whole, satisfactory and sufficient for the purpose at hand. Of these, only the last need concern us. Prof. Ladd denies that we have at present any means of explaining the working of the nervous system on strictly mechanical grounds, or, as he says, a precise mechanical theory of the nervous system is at present an impossibility. He does not deny "the machine-like nature of much of the structure and movement of the human body," and it is only on ultimate analysis that "we must, after the discussion of all analogies, resort again to the unknown molecular constitution and properties of the substance of the nerve as being *sui generis* for an explanation of its peculiar physiological properties." That is, once given the nerve and its ganglion cell, all else follows in proper succession, and in accordance with the laws of molecular physics; but of nerve action itself we can frame no proper mechanical theory.

Part II. deals with the "Correlations of the Nervous Mechanism and the Mind," and is introduced by a full discussion of the localization of cerebral function. The safe middle ground is the one chosen, and the conclusions are conservative and, on the whole, just. As an example of praiseworthy conservatism, we may cite (p. 260): "The rash confidence with which the brain of the monkey has been mapped out in detail, and human pathology ransacked with the purpose of finding some warrant for copying upon the brain of the human species, cannot be too carefully avoided." It is unnecessary to add after this that Ferrier's results are criticised with much severity, and Ladd concludes, that of "the large number of so-called centres pointed out by Ferrier and others, . . . fully half cannot be regarded as having anything like a demonstrable character."

As regards the motor centres, Ladd thinks that "the convolutions on either side of the fissure of Rolando (the *gyri centrales anterior* and *posterior*) and the connected lobule of the median surface of the brain (the *lobulus paracentalis*) are in the highest degree especially connected with the motion of the extremities of the body; that adjacent parts of the frontal and parietal lobes are thus connected in a less degree; that the cortical region for the arms lies, on the whole, anterior to that of the legs; and that, probably, the region for the hand is "near the middle part of the front central convolution, and that for the tongue where the mid-

dle and lower frontal convolutions meet the central. More precise localization of the functions of man must as yet be made with a lower degree of confidence. Beyond these general statements lies the undefined field of conjecture," p. 282. While admitting the importance of the occipital lobes for vision, yet apparently Prof. Ladd has had no knowledge of the more recent pathological contributions to this subject, and especially the articles by Seguin on "Hemianopsia." But if physiological psychology may not claim that the study of the localization of function is a field peculiarly its own, it may turn to the vast field of psycho-physics and psychometry with full assurance that its claims here will be undisputed, and perhaps the experimental study of the sensations has achieved nothing of more importance than the new interpretations that have been given to the law of the specific energy of the nerves. Vision and sound have already received their due share of attention, but it has remained for the last two or three years to develop a distinct "skin psychology." To the brilliant results achieved in this field, Prof. Ladd does full justice, and heat-spots, cold-spots, and pressure-spots show the increasing possibility of differentiation of function and of making new distinctions in the qualities of sensation. But the discovery of these localized sense perceptions is but a very small part of the skin psychology, and the study of touch, of sensation circles, motor sensations, etc., have alike been prolific in increasing the knowledge of our sensational life. Of the results of all these researches and of the many allied ones in optics, acoustics, and psycho-physic time, Prof. Ladd has given us fair and accurate accounts, and his conclusions are usually unexceptional. Any discussion of Weber's law must necessarily be unsatisfactory at present, and we can certainly agree that "the experimental proof of Weber's law is as yet too incomplete to make us ready to accept it as an ultimate psycho-physical principle." Certainly much confusion with but little commensurate gain has resulted from its introduction into psychology, and although we may accept Ladd's general statement of it, that *every mental state has its value determined*, both as respects its quality and so-called quantity, *by its relations to the other states*, yet as regards any strictly mathematical mould for our sensations we may well say with him, "why the quantitative relations of body and mind should be such, and such only, that a geometrical series of changes in the one should invariably be represented by an arithmetical series of changes in the other must indeed remain an ultimate mystery" (p. 380).

Into his discussion of the "Time Relations of Mental Phenomena and the Feelings of Emotions," we may not enter, but a few words at least are demanded on "Attention and the Physical Basis of Volition."

*There is no special organ of the will*, is the somewhat abrupt conclusion of Prof. Ladd. Were we to dispute this with equal abruptness, we would say, *the organ of the will is the striped muscu-*

*lar fibre*. In other words, we do not find any adequate presentations of the rôle of the voluntary muscles in volition nor of the experimental researches on this subject.

Prof. Ladd has reserved the concluding hundred pages for a discussion of the nature of the mind. Although he nowhere attempts to conceal his contempt for a "psychology without a soul," he does not introduce any metaphysical discussions into the body of the book, but reserves for the end his conclusions and judgments of the claims of physiological psychology. His judgment is that physiological psychology may legitimately undertake to answer the question, What is mind? and his answer is as follows: *The subject of all the states of consciousness is a real unit-being called mind, which is of non-material nature, and acts and develops according to laws of its own, but is specially correlated material molecules and masses forming the substance of the brain.*

To discuss this would lead us too far afield; but we may confidently say that it is a definition with which no neurologist or alienist will agree. Perhaps the time may come when no one will attempt to define mind without taking into consideration the facts of *morbid* psychology; but until that time we shall probably look in vain for a satisfactory definition.

Prof. Ladd, then, has supplied a distinct and long-felt want in putting forth a volume that covers so well the immense field of psycho-physical investigation. If it seems somewhat "scrappy" and somewhat indiscriminating in the relative importance of certain psycho-physic subjects, every concession must be made to a first attempt in a wholly new field. To the alienist or neurologist it will prove of great service in presenting a mass of facts hitherto scattered through the literatures of many different subjects. As an earnest of what the future may bring forth, the volume should be given a cordial welcome.

WILLIAM NOYES.

## Society Proceedings.

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### NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, April 7th, 1887.*

*The President, C. L. DANA, M.D., in the Chair.*

REPORT OF A CASE OF SARCOMA OF THE OCCIPITAL LOBE,  
CAUSING HEMIANOPSIA, REMOVED BY OPERATION.

By DR. W. R. BIRDSALL and DR. R. F. WEIR.<sup>1</sup>

REPORT OF A CASE OF CYST OF THE BRAIN, WITH OPERATION.

DR. GRAEME M. HAMMOND related the case. The patient, a married woman, complained of severe pain an inch above the right ear, and had left hemiplegia. At the time her symptoms began, she was about twenty-nine years of age; she had had four children. During the progress of her disease she had a fifth child. All were strong and well. Her sickness lasted about two years and a half. It commenced by sudden loss of consciousness and convulsion limited to the left side. The left side of the face became, and remained, paralyzed. About a year later she noticed gradual loss of power in the left arm; things dropped from the hand. At the end of another year, the left arm was completely paralyzed. She then noticed increasing weakness in the left leg. When Dr. Hammond first saw her, she was able to stand and walk slowly. While the paralysis was extending, she had four or five epileptic seizures, confined to the left side. Headache developed about the time the legs became affected, grew more constant, and was described as agonizing.

She denied syphilis. Physical examination showed loss of motion on the left side of the face, tongue, and soft velum; of the left arm, and partial loss of motion in the left leg. There was no

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<sup>1</sup> See this JOURNAL for May, 1887.

disturbance of sensibility of any kind. The reflexes were exaggerated on both sides. Sight, hearing, smell, and taste were normal. Choked disc on both sides was observed at a subsequent examination.

Under treatment, the headache was controlled to a considerable extent. Dr. Hammond's diagnosis was probable cerebral tumor, involving the cortical or subcortical substance of the motor centres. An operation was refused. The patient grew worse until the 20th of March, 1887, when her husband asked that the operation be performed. Dr. Spitzka then saw the patient with him, and made two examinations. He rather coincided in the diagnosis. The operation was performed in the hospital last Wednesday. Dr. M. Josiah Roberts assisted at the operation, removing the portion of skull by his electro-osteotome. Four buttons were removed by the electro-trephine; they were connected by straight lines made with the circular saw. The diameter of the opening was over two inches. A crucial incision was made through the dura. A hypodermic needle was introduced in different directions, but no fluid was withdrawn. The dura was closed. A drainage-tube was introduced, the skin flaps were sewed up, the patient was put to bed. She lived only twenty-one hours, remaining unconscious after the operation. Prior to the operation, she had become completely paralyzed in the left lower limb; she had delusions and hallucinations; she talked incoherently; bed-sores developed.

The autopsy showed little hernial protrusion at the wound; the cortical substance here was thin. Incision through the motor region revealed three cysts in a line, deep in the white substance. The fluid in the cysts has been only partially examined: it was serum, and contained broken-down brain substance. The cysts were close together, and in a position to affect the face, arm, and leg centres. He could not explain why the syringe failed to bring forth fluid, unless it was that the cysts were too deeply situated or the needle passed between them. He added to the clinical history that the head was drawn to the right side the last three days of life.

#### DISCUSSION.

DR. E. C. SEGUIN was partly responsible for the operation in the first case, but it seemed the patient would not live more than two or three months without it. He expected to find a large tumor, but was somewhat surprised to see it encapsulated and non-parenchymatous. During the early history of the case the



symptoms pointed to destruction of brain tissue. He asked Dr. Hammond whether the sensory or motor symptoms determined the seat of his operation.

DR. HAMMOND replied that the headache corresponded to the centre for the motor symptoms and the seat of the operation.

DR. SEGUIN added that the seat of pain would be a very uncertain indication for the seat of the operation. In some cases of cerebellar tumor, for instance, the pain had been mostly frontal.

DR. E. C. SPITZKA, referring to the case reported by Dr. Birdsall and Dr. Weir, said that an artery, large enough to cause fatal hemorrhage, entered the gray and white substance of the right occipital lobe. It had been overlooked in many text-books.

DR. ROBERTS explained how the circular saw could be used without injury to the brain; and the operation of the electro-osteotome.

DR. STARR suggested the desirability of an analysis of reported cases of cerebral tumors for the purpose of determining their rapidity of growth and size.

DR. R. L. PARSONS read a paper entitled:

NOMENCLATURE IN PSYCHIATRY. MONOMANIA OR OLIGOMANIA, WHICH? PARANOIA, WHAT? (See this JOURNAL, April, 1887.)

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*Stated Meeting, May 3d, 1887.*

CHARLES L. DANA, M.D., *President, in the Chair.*

The election of officers for the ensuing year resulted as follows:

For President, C. L. Dana, M.D.; for first Vice-President, W. R. Birdsall, M.D.; for second Vice-President, M. A. Starr, M.D.; for Recording Secretary, G. W. Jacoby, M.D.; for Corresponding Secretary, W. M. Leszynsky, M.D.; for Treasurer, E. C. Harwood, M.D.; for Councillors, E. D. Fisher, M.D., B. Sachs, M.D., L. Weber, M.D., E. C. Seguin, M.D., and G. M. Hammond, M.D.

NOMENCLATURE IN PSYCHIATRY. MONOMANIA OR OLIGOMANIA, WHICH? PARANOIA, WHAT?

The discussion of this paper by DR. R. L. PARSONS, read at the last meeting of the Society, was taken up.

DR. KELLOGG said he was not well informed of the contents of Dr. Parson's paper, but he would say that it seemed to him the term monomania had come into so general use in literature

both medical and medico-legal, that it would be very difficult to get rid of it. It was very easy to use new terms, and perhaps etymologically they might be more appropriate than the old, but there was a practical use of the word monomania, and it was very difficult to find another which would fill its place. Monomania had been employed to indicate many different conditions of mental disease. If we could limit the clinical group of mental symptoms, we might then suggest more exact terms. There was a group of clinical symptoms associated with certain neuroses, such as epilepsy and chorea, also sometimes associated with toxic states, as in alcoholism, or with diathetic states, as syphilis or tuberculosis, which took the form of a fixed delusion. By some authors such symptoms had been classed under the term monomania. Other authors had applied this term to conditions of perverted emotions. What one term would better indicate all these conditions than the word monomania? He thought none. All that he could do would be to single out the several clinical groups of symptoms included under the term monomania, and apply to each group a distinct term. He thought, however, the generic term monomania would remain. At any rate, he could see no advantage in seeking for a term which would replace it. If Dr. Parsons wished to designate one distinct group of symptoms from among several which were now included in the term monomania, by the use of the term oligomania he might agree with him, but he did not think any advantage would be derived from substituting the latter term for the former.

DR. W. R. BIRDSALL had listened with interest to Dr. Parsons' paper on "Nomenclature in Psychiatry," because of the importance and difficulty of the subject, but the impression left upon his mind was that we were not much better off with the terms coined in recent times. He must certainly agree with the author regarding the indefiniteness with which the term monomania had been employed, but he agreed with Dr. Kellogg that in its generic sense it was pretty well understood. There was, however, a popular misconception regarding its meaning which he thought constituted a strong objection to its use. Dr. Birdsall had come to use the term paranoia as a substitute for that of monomania, with satisfaction to his own mind. The objection to it mentioned by Dr. Parsons, that it did not mean much if anything, in Dr. Birdsall's opinion constituted one of its merits. Monomania was so definite as to be liable to misconstruction. In reconstructing nomencla-

ture, he sometimes thought it was a misfortune that we could not fall back on Choctaw, instead of being compelled to resort to Greek or Latin. There was a certain disadvantage in having to resort to a compound word to express a group of symptoms or type of disease; it often failed to describe anything clearly. He believed, with Dr. Kellogg, that unless the symptoms included in the general term monomania were subdivided into groups, and an appropriate term for each group was found, little advantage would be derived by substituting a single new term.

DR. B. SACHS objected to the term monomania for two reasons: first, its etymological meaning was different from its accepted meaning in the nomenclature of psychiatry; second, in accepting its use, we implied our approval of the old division into *monomanias*, and thus lost sight of the true character of serious mental troubles. He thought the term oligomania was less objectionable than that of monomania, but he did not believe there was any absolute need of either term at the present time. He had not been compelled to make a diagnosis of monomania for several years. In general, he agreed with Dr. Birdsall's remarks on paranoia, but he thought also that the term primary insanity would be little liable to misconstruction, and might on some grounds be preferred either to paranoia or monomania. He would be in favor of using the term paranoia or primary insanity with such qualifying phrase as to the character of delusions, etc., as might be necessary in different cases.

THE PRESIDENT thought Dr. Parsons deserved credit for his paper, as there was certainly need of improvement in our nomenclature. If the paper had been read five years ago, the term oligomania would probably have stood a better chance for adoption. But it had now become fashionable to use the term paranoia, and he, like Dr. Birdsall, felt a certain amount of mental satisfaction in its use. He had some time since come to the conclusion that any attempt to reform nomenclature, if by a single individual, would be utterly useless. Such questions must be referred to conventions. One man could do nothing unless he discovered some fundamental principle on which to base a nomenclature which would prove superior to that now in use.

DR. PARSONS thanked the members for the adverse as well as the favorable criticisms of his paper, and in reply repeated certain statements made in his paper. He did not believe that any convention of men could alter a nomenclature. Whether or not a

given term would be adopted depended much upon accidental circumstances. He looked for advances in nomenclature rather from individual effort. He had not found it necessary to use the term monomania; the term partial insanity was more applicable in many cases.

#### CASE OF SUPPOSED CEREBRAL TUMOR—PARTIAL REMOVAL.

DR. S. N. LEO read the history of a case of cerebral tumor occurring in a heavy German woman who had before its appearance sustained an injury of the skull where the tumor afterward developed. During the growth of the tumor, the patient had consulted several well-known physicians, all of whom were of the opinion that an operation should be performed. Some thought it was a wen of the scalp. The symptoms becoming more serious, she finally asked Dr. Leo to perform the operation. He was assisted by Dr. Harwood and Dr. Guleke. Strict antisepsis was observed. As the operation proceeded, it was found that the tumor extended within the cranium, involving the dura mater at the longitudinal sinus, and apparently dipping deeply into the brain substance. These facts, in addition to the patient's bad condition from loss of blood, led him to desist from further procedure after cutting off that portion of the tumor external to the cranial vault. He thought the patient would not have lived so long (over a month), had not the operation been performed. Unfortunately, an autopsy was not allowed. The tumor was a sarcoma. He added that the patient's condition had been rendered more serious by cardiac disease.

DR. L. WEBER regretted that the title of the paper on the cards of announcement had been "Report of a Case of Cerebral Tumor Successfully Removed." He also regretted that there had not been a post-mortem examination.

DR. B. SACHS then read a paper entitled:

#### NOTES ON THE CAUSE AND TREATMENT OF FUNCTIONAL INSOMNIA.

Under this term he included cases of insomnia, pure and simple occurring in persons of the neurasthenic habit. He preferred to say neurasthenic rather than hysterical, for in his experience actual insomnia is less frequent in truly hysterical patients than in those suffering from cerebral or spinal neurasthenia. A number of typical cases were given. In attempting to explain

these cases, and work done by physiologists on this head, the reader concluded that in the majority of cases we had good evidence of disturbances in cerebral circulation. And as Mosso had found in animals that increased activity of cerebral circulation was accompanied by deficient circulation in the peripheral organs, so in many cases of chronic insomnia, cold extremities, pallor of the skin, scanty uterine flow pointed to deficient peripheral circulation, and in many of these cases there were weak heart and weak pulse. Special attention was called to the simultaneous occurrence of insomnia and headache, and that in these cases the headache was, as a rule, of the *paralytic* migraine type. Cases were quoted in evidence.

The treatment in cases of migraine and insomnia was similar in many respects, and proved effectual in both conditions.

As regards treatment, the point the author of the paper wished particularly to insist on was that continued hypnotic medication was worse than useless; that the good results obtained by him were due to close attention paid to matters of general *régime*; to the treatment of an existing anæmia; to the strengthening of the force of the heart's action by cold douches, by the regulation of exercise, and the methodical performance of definite forms of active physical exercise, such as riding, rowing, and mountain climbing. Hypnotics were of use only at the outset of treatment; among these the reader recommended chloral and bromides to be given at night, the bromides alone, amorphous hyoscyamia, urethan (2.0), and paraldehyde (4.0-6.0 in wine); but that these hypnotics were to be withdrawn as soon as a slight improvement in sleep was noticeable, and from that time onward general treatment was to be pushed vigorously.

DR. FISHER thought a very common cause of insomnia was anæmia, and he had seen considerable success in its treatment by cod-liver oil, cream, and articles intended to improve nutrition. In some of the cases, ordinary hypnotics had been administered without any avail. The patients might have the appearance of being well nourished while they were really anæmic. The mineral tonics were, as a rule, indicated.

DR. GEORGE W. JACOBY thought the paper was an exceedingly important one, especially in that it called attention to the fact that many cases of insomnia could be cured by rational measures alone, without the use of any medicines whatever. He agreed with the author that the cases must be individualized, and thus

the cause of the wakefulness might be discovered. He thought that in the majority of cases the cause would be found to lie in the circulation; not always in anæmia, but frequently hyperæmia. Cure the cause and we would cure the sleeplessness. But that which would cure anæmia in one case would not cure it in another. Active and passive exercise, particularly active exercise, were of benefit. For patients who could not go out, the muscle beater was very useful. While he had not much faith in static electricity in the treatment of insomnia, he cited one case in particular in which the physician who applied it for another purpose to one of his patients, himself became sleepy under its influence. Perhaps the production of ozone by the instrument was the cause of this sleepiness; for it was well known that when we went into an atmosphere of ozone we were likely to become sleepy.

DR. V. P. GIBNEY had noticed that static electricity tended to produce the sleep state. It was one of these things they had found static electricity good for at the hospital with which he was formerly connected.

DR. W. R. BIRDSALL thought, as did Dr. Sachs, that we must adopt hygienic rather than purely medicinal measures for the cure of insomnia, but we were occasionally forced, as the author had said, to resort to some drug for temporary relief. For this purpose he had obtained benefit without injurious effects, such as sometimes came from bromide, hydrate of chloral, etc., from a drug first recommended to him by Dr. Seguin, namely, conium. This given in large doses, fifteen or twenty drops or more of the fluid extract, had in his hands been beneficial. He had continued its use two or three months without deleterious results.

DR. G. M. HAMMOND thought fully eighty per cent of all his patients were similar to those described in the paper by Dr. Sachs—persons suffering from insomnia, mental anxiety, etc. In the large majority of the cases, he thought it was due to hyperæmia of more or less limited areas of the brain. When the patients did sleep, they had unpleasant dreams. They were also frequently sufferers from dyspepsia, constipation, spots before the eyes, noises in the ears, sometimes hallucinations connected with various senses, and coldness of the extremities. It was rare for such patients to go away without being cured, but if they subjected themselves to the same causes, the condition returned. He used bromides, and stuck to them right through the disease. He gave only ten or fifteen grains three times a day, and also gave fluid extract of ergot. He

applied static electricity and dry cups to the back of the neck, and regulated the sleeping hours.

DR. LESZYNSKY was rather surprised, in view of a recent discussion before the Society, to hear Dr. Sachs speak of the use of hyoscyamine as a hypnotic. It was a mistake to rely upon large doses of bromides given at night. There was an objection to their use in ladies, because of the bad odor which they gave the breath. He had not been able to discover any peculiarity in the circulation of the retina in these cases.

DR. L. WEBER said that since he had adopted the treatment recommended by Dr. W. A. Hammond, and just described by Dr. G. M. Hammond, he had obtained the best results in suitable cases for this mode of treatment. But in other cases the bromides might cause excitement instead of aiding sleep. When there was gastro-intestinal disorder, he added to the treatment calomel with benefit.

DR. LESZYNSKY referred to a remark by Dr. Birdsall concerning the use of a mustard sinapism, or other cutaneous irritant, and says that Dr. W. H. Thomson had called attention to the beneficial effects of Cayenne pepper, etc., to the surface of the body some years ago.

THE PRESIDENT had found the warm bath a very valuable measure in many cases; in mild cases of insomnia the cold douche down the back and massage had proven useful. Binz had discovered that ozone has a hypnotic influence. Lupulin had been of benefit in the insomnia of old people, and lavender in some cases in which the stimulus of alcohol or warm food had failed.

DR. SACHS objected to the use of bromides, particularly in small doses, more than to anything else in the treatment of the class of cases under discussion, namely, those of insomnia in neurasthenic subjects. It was likely to do more harm than good. The testimony at the discussion referred to by Dr. Leszynsky was not against amorphous hyoscymia, but against the crystalline form.

THE PRESIDENT exhibited

AN APPARATUS FOR THE RELIEF OF WRITER'S CRAMP,

called the "Kaligraph" by its inventor, the late Mr. Charles Thurber. It consisted of an iron framework, to which was attached a series of levers so arranged that by making large characters at one angle the characters were reproduced in ordinary size at the opposite angle. It was, in fact, a kind of reversed panta-

graph. Dr. Dana said that all writer's-cramp instruments were based on the principle of resting the groups of muscles most used and throwing the work upon other groups. The kaligraph fulfilled these indications better than any other instrument with which he was familiar. The objections to it were that it was cumbersome and expensive. The speaker showed cuts of all the various forms of instruments for writer's cramp (10 in all) which he had been able to collect. The kaligraph had been in practical use for thirty years, but it was very little known. It had enabled its inventor, who suffered extremely from the cramp, to write with comfort. He was informed that Charles Dickens had possessed and used one.

DR. G. W. JACOBY thought this instrument was only palliative while Nussbaum's was also curative, and could be carried with one. It compelled the writer to use the abductors.

THE PRESIDENT replied that an instrument calling into play another group of muscles of the hand would cause those to be affected after a time.

DR. BIRDSALL thought writer's cramp was due to cerebral fatigue rather than to muscular fatigue, and that instruments for overcoming it could be of only limited benefit.



## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Regular Meeting, February 28th, 1887.*

*Vice-President, DR. CHARLES K. MILLS, in the Chair.*

I. N. KERLIN, M.D., read the following paper on

### MORAL IMBECILITY.

I present short histories of several children—the first, presented by my friend Dr. Carson, of Syracuse, New York, illustrative of the incipient prostitute whose mental incapacity should be her protection; the second case, the incipient burglar; third, a hereditary religious hypocrite and egotist who, if not permanently sequestered, will fill a dramatic if not an awful rôle in crime; fourth, a confirmed juvenile tramp and incipient confidence man.

*Case I.*—Tom McK., aged 12, admitted November 9th, 1877. When first brought to our knowledge he was described as an incorrigible boy, who had been passed from one county home to another, through a juvenile reformatory, and at last, to prevent his own self-destruction, because of his propensity to climb the rods and water-spouts of the refuge and to ramble dangerously over its roofings, he was locked in a secure room. Excepting his under stature, nervous manner, and glittering eyes, there was nothing in the aspect of the pale-faced boy to suggest any unlikeness to normal boyhood. Indeed, his aptness in language, both usual and profane, suggested precocity. He was but little more backward in his studies than would be any neglected boy; full of mischief and deceit, he had the usual indifference of a bad boy to punishment morbidly increased. There seemed to be a moral hebetude and a causeless wilfulness, that had taken the place of the fretfulness, kickings, and bitings of his earlier childhood.

The boy promptly responded to school instruction; is described in the earlier weeks as a "nice little boy, who I think will learn."

Favorable reports continue to be made of his intellectual progress during the next two years. In 1882 he is spoken of by an

indulgent teacher as "a healthy, strong, and fine-looking boy, manly and frank;" but notwithstanding these favorable reports, there were seizures of irritability and excitement occurring periodically, and of increasing intensity the longer he remained with us.

The inventions of his cruel nature, directed against child and brute alike, cannot be detailed. The vindictiveness with which he followed up any one against whom might be developed a temporary suspicion was satanic. The secrecy with which he could lay plots to steal from his companions, and the adroitness with which he covered up his tracks, were those of an expert.

The period of his discharge from the institution had arrived, and Tom was restless to return to his people, and they equally determined to have him do so. Six months after his discharge, I was importuned to testify in court for his release from imprisonment. In the brief period following his dismissal from the institution, he had already been found guilty of two or more petty burglaries. Benevolent friends had discovered him in prison, freely confessing his offenses, and weepingly imploring to be sent back again to the institution. The crimes seemed to be unaccompanied with sufficient motive to make them heinous.

After a thorough and painful review of his whole life with us and the discomforts that must follow his readmission, I was compelled to advise that the course of the law be not interfered with, but urging that an intermediary, such as that at Elmira, should enter the scheme of our penal treatment.

This incorrigible moral imbecile was the son of a man of most abandoned character, who in his paroxysms of drunkenness was a savage. I recently discovered his mother in the insane department of a county house, one of the saddest of demented. The boy's conception, birth, and childhood—his whole history—had been laid in physical disorder, fright, and dissoluteness.

*Case II.*—G. L., admitted July 12th, 1883; a sweet-faced, intelligent, lovely boy, whose gentle manners instinctively won confidence, and made his new-found friends at the institution believe that the boy had been improperly consigned to our care by a mother too eager to relieve herself of her proper relations to her own child.

The mother's description of this lad (she withheld some points we afterwards discovered) is so good that I quote here at length. "He is fourteen years old; large, healthy, well formed; kind-hearted, quick-tempered, very dull of comprehension; seems to lack the

power of either continued study or labor; full of good resolutions and promises that are seldom kept; determined to see the inside of anything at any cost; fond of working with any kind of tool; fond of little children, and a lover of horses and dogs. A hearty hater of books; can scarcely read, though he has had at least ten months' term at school. Will listen to verbal instruction and ask questions from morning until night. The average child of eight years has better reasoning powers than he." "He is not malicious, but is destructive; is reckless of danger, not regarding consequences, and I am at a loss to decide whether he cannot or will not weigh them. He will both lie and steal, but neither are common practices. When I have found he was guilty of either, I have always found it was done on the impulse of the moment through sudden temptation. *He will not confess.*"

This interesting boy took an upper place at once in our community. Located in one of the best rooms, associated with the best group of boys, his face shining with sweetness, Guy went along swimmingly for nearly three months. Little meannesses were complained of by his associates, and we found that he had few friends among the boys. Notwithstanding his loveliness to us, he was an object of aversion to them. Instances of cruelty to the feebler children; petty pilferings from his associates' boxes, opened by ingeniously twisted hair-pins, made numerous counts against him, of which he was generally forgiven because of the lack of clearness in the testimony and his adroit evasions. He devoutly knelt at his bedside every night, was thoroughly up in Romans, Timothy, and the Beatitudes, and stood well in our Bible class. His trouble with the boys found commiseration from our tender-hearted ladies, so that while in disfavor in the dormitories he was in high favor with the court.

On a quiet Sunday afternoon, when the children were scattered in their various clubs in distant parts of the grounds, excepting that to which G. belonged, our carpenter shop was discovered in flames. The locked door had been opened by prying, a window was noticed to be partially elevated, and marks of violence upon a tool chest in which matches were usually kept by the carpenter. Immediately after the fire an attempt was made to sift the testimony of eight or ten feeble-minded boys, the substance of which pointed to one of two or three boys as possibly involved in the incendiarism.

This preliminary examination resulted in placing G. in confine-

ment, with the hope of eliciting from him such confession as might corroborate the circumstantial evidence that pointed to him.

Through three long weeks this sweet-faced, praying, pleading boy held out against all kindness, all persuasion, and we were well-nigh giving up the task, when finally the following letter addressed to his mother passed through our office :

"DEAR MOTHER.—I cannot be satisfied until I tell you what I have done. I am very sorry to tell you of it, but it is better for me to tell it myself. On last Sunday I thought I would like to see how the fire-machines were used, and I got into the carpenter shop, took a match out of the cupboard, and set the shavings on fire. When the blaze got about a foot high, I hallooed 'fire' before it got too big. I cannot rest until I tell you. Every one treats me too kind to do anything like that in spite to any one. I cannot tell you how I feel, my heart is like a lump of lead. I am so afraid that you will think I am never going to become a good man. I have asked the Lord to forgive me, and I know He will if I obey Him. I have learned the fifth chapter of Romans, and I am going to be a good boy after this," etc.

I found by interrogating the police authorities of his town that he had, prior to his admission to our institution, been shut up for petty larceny, and a late admission came from his mother that he had fired her own house. With these facts before me, I placed him in the House of Refuge, where, two weeks afterwards, it was sincerely believed that the boy had been wrongfully committed, so well had he played his rôle of sweetness, and that the confession had been a forced one under the distress of confinement.

The etiological history of Guy is somewhat interesting. The parental grandmother is described as a fanatic on the subject of religion ; the eldest son of this grandmother thinks that he lives without sin, and his brother (this boy's father) is of the same belief, and has been at all times deranged on the subject of religion. The mother's testimony is as follows : "Practically my son might as well have no father, whose only faculties are building engines and studying the Bible. He passes through the world neither seeing, hearing, nor feeling anything that is not directly connected with either of these his two hobbies. Notwithstanding his devotion to Scripture, he is subject to attacks of brutal anger."

*Case III.*—Frank L. is thus described on his admission to our school, March 24th, 1880, by the teacher in charge : "He is 12 years old, has attended school in winter for the last two or three

years, and in summer worked on a farm. Notwithstanding his limited advantages, he can read in the American Third Reader and write an intelligible letter ; adds eights, sixes, and fives ; can write short sentences ; knows the primary and secondary colors ; can tell time ; draws a little. In form, knows the square, oblong, and circle. Smiles a great deal ; is a little deaf. In April he is described as showing much interest in a few simple lessons in botany, writes short letters, very well expressed. He is a good boy in school."

Scarcely a symptom of intellectual or moral infirmity had been discerned in this interesting boy when, without known reason, he eloped, which threw our family into great distress, relieved only when writing the father upon the subject, who replied, "I can take no steps toward his recovery ; I have already spent hundreds of dollars in bringing him back from just such escapades."

The boy had been committed to us by Judge Ross, of an adjoining county, who found this blue-eyed, sweet-faced lad in the cell of their county prison, beating his head violently against the wall and asserting that he "meant to make himself like his mother, who was already crazy." The sympathetic judge recognized the imbecility of the boy, and refused to pass judgment upon the trivial offense for which he had been arrested.

A few months after his elopement, a newspaper sent to us from Escanaba, Michigan, related the story of a boy who had appeared in that town and who had excited a very great interest.

The boy had been found standing on the shore of the lake, weeping bitterly and explaining to citizens that his father had just thrown himself overboard and drowned. Boat-hooks and fishing-nets were brought into requisition, and for many hours there was an earnest endeavor to recover the missing father, when a more reflecting citizen, upon close interrogation of the boy, discovered that the story was a hoax. The editor of the morning paper took a friendly interest in the boy and carried him to his office, and explained the case the next morning at great length in a sensational column to be one of insanity from exposure and hardship. The evening paper of a political rival came out with a column protesting that the little fellow was an incorrigible waif from one of the Eastern refuges, and had hoaxed its half-witted contemporary by playing off the insanity dodge.

It proved to be our Frank, for among conflicting statements he had said that he had an uncle near Media, Pennsylvania, which

resulted in the return of the boy to our institution, but for a brief season only; for, after six or eight months of diligent attention in school and workshops, and seemingly contented and happy, he suddenly, without known cause, left for parts unknown. After many months, he was reported by the police authorities of Chicago, and through the courtesy of these public-spirited gentlemen, and of our own police force in Philadelphia, Frank was returned, to come into the office with the same blue-eyed confidence, to settle again at the harness, and with the keenest adaptation to resume his alto horn in the band. Again, without any reason for it that could be ascertained, suddenly and as certainly as before he again eloped, and is at present somewhere within fifty miles of the institution, occasionally appearing to acquaintances quite near us, but mysteriously fading away.

#### DISCUSSION.

DR. H. C. WOOD.—Whether we call these cases examples of moral imbecility or give them some other name, we must recognize that there is a depravity which is the result of heredity or some imperfect development of the nervous system. I recently had under my notice a case which parallels those reported by Dr. Kerlin. A woman belonging to a respectable family went astray at a very early age and became the mistress of a married man, bore eight children, and died a drunkard in the gutter at the age of twenty-eight. Of the eight children, all, with the exception of one, perished in infancy. At the death of the mother, this child, a very young girl, was taken by a friend, who endeavored to train her in a respectable manner. The attempt seemed to be successful until the age of thirteen was reached, when, three months before the appearance of menstruation, she ran away and was found in a house of prostitution. Since then her history has been a constant effort to save her and a constant return to bad sexual habits. It finally became necessary to place her in the House of Refuge. There was no tendency to drink in this instance, so that the love of liquor had no part in the precocious, overpowering sexual desires.

DR. E. N. BRUSH.—I was much interested in the graphic description of these cases. I had the opportunity of seeing one of them at the institution at Elwyn. The history of the young woman brings to mind a case which had gone, as Dr. Kerlin says they may do, a little farther. The girl not only became a public

prostitute, but had passed into a condition of marked insanity. The mental disturbance was as much in the line of moral obliquity as anything else. She was very active in causing mischief among the attendants and other patients at the asylum. At one of the visits of a committee of inspection she trumped up a story that that the engineer, the apothecary, and one of the physicians had come to her room at night and violated her person. She got one old gentleman very much interested in her case, but before he got through with his investigation disgusted him by making a similar charge against him.

DR. CHARLES K. MILLS.—I believe that the term moral imbecility is a good one to apply to a certain line of cases, as it has a distinctive meaning. From my experience outside of institutions I could give at least a score of cases similar to those described by Dr. Kerlin. Some of these cases have been in asylums, some have been in prisons or houses of refuge, and some have been in both. I recall one case, now in the Eastern Penitentiary, which has been in three asylums and in three or four penal institutions. A case recently sent to the Pennsylvania Hospital for the Insane by Dr. Wood and myself presents some of the elements which have been mentioned by Dr. Kerlin—some of the peculiarities seen in the first case reported by him, although not to the same degree. In addition, she had actual spells of mental excitement, possibly a mild form of mania. In the case of a boy recently seen, as in this case, there was a distinct history of neurotic conditions and of insanity in other members of the family. Almost all cases of this kind show a marked element of heredity.

As I have stated, I am a believer in moral insanity, as I understand the term, although I do not consider this term a good one, especially for medico-legal purposes. I have been asked on other occasions to produce the cases. Dr. Kerlin has produced four to-night. I think that the members of this Society could bring forward a large number of other cases. The question is not whether or not these subjects have any intellectual aberration at all, but whether or not the moral aberration vastly predominates. If so, the cases should be classed under moral imbecility or moral insanity, whether or not these terms are the best for technical purposes.

DR. JAMES HENDRIE LLOYD.—I unfortunately heard only a small portion of Dr. Kerlin's paper. I think that in the discussion of this question, especially in its practical bearing upon the cases

that come before the courts, medical experts are in danger of confounding depravity with insanity. There is a decided distinction between these two. I have always maintained that the mere metaphysical abstraction of moral insanity was an unphilosophical one. Practically we never find a case of a pure moral lesion. There is always an intellectual defect, and frequently a marked congenital defect of the whole brain organization. We unfortunately know very little of the physical basis of thought, and, until we do know more about this, it seems to me that it is futile to try to make abstract distinctions between moral insanity and depravity. This tendency was probably inherited from the pseudo-psychologists who combatted the idea of Locke in regard to the action of the human understanding. I myself do not believe in true moral insanity. In the last case reported, if there were no evidence of mental defect, it seems to me a misnomer to call the boy an imbecile. If the boy was full of depravity, and if he had committed a crime, it would be difficult to acquit him before an intelligent jury. I suppose Dr. Kerlin would not claim that he was without intellectual defect.

DR. FRANCIS X. DERCUM.—I think that Dr. Lloyd is correct as regards the unphilosophical character of the term moral imbecility or moral insanity, as used in the *abstract* sense. At the same time I can conceive of cases such as Dr. Kerlin has reported. As man ascended from the savage state, the moral faculties were the last developed. Their highest development is inseparably connected with the more intimate social relations demanded by civilization, and is therefore of comparative recent origin. Now, I can conceive of a child in whom the reasoning powers and the other faculties were developed to an average degree, just as in the savage, and in whom the moral faculties were undeveloped. I believe that in these cases there is a structural and incurable defect—a morphological defect.

DR. HARRIET BROOKE.—There is in the Woman's Insane Department of the Philadelphia Hospital a young woman, sixteen years of age. She is intellectually very bright. She works efficiently about the ward, and visitors are surprised to learn that she is insane. She has, however, a most violent temper, she steals, she is untruthful, she is cruel, and, when she is crossed, becomes so violent that she takes anything within her reach and throws it at those around her. At such times she is perfectly irrational. When brought to the hospital by her mother and her



sister, the latter volunteered the statement that on the father's side there was no insanity, but that the mother of the girl was the only one of the mother's family that was not insane. The sister had herself been insane twice. This case seems to belong to this group of moral insanity.

Dr. ISAAC N. KERLIN.—We will be more grateful to Dr. Lloyd if for the terms moral insanity and moral imbecility he will give us expressions defining any better the conditions which we find in a large percentage of so-called feeble-minded persons in many of the commitments to houses of refuge, and in a larger proportion still of those found in such institutions as the reformatory at Elmira, New York.

Is not the difference between those who argue for and against moral insanity a disagreement about a name, rather than about the existence of a condition accepted by most Germans and many English writers on psychiatry, and supported by Ray, Spitzka, Bannister, and many others in America?

During the past few years, there seems to be a growing advance in sentiment among our profession and in the public mind with reference to this subject. Seven years ago, a paper read at Lincoln, Illinois, reporting several cases of moral imbecility under the title "Juvenile Insanity," was so thoroughly misunderstood that a discussion followed showing that the gentlemen present had applied the phenomena of *excitable idiocy* to the suggestions of the paper. The idea of a congenital incapacity to receive and act upon moral impressions in any other than the lowest grades of intellectual idiocy was not comprehended. Two years ago, I wrote to the non-medical superintendent of one of our houses of refuge, sketching a few of our moral imbeciles, and asking if there were cases corresponding with these in that institution. The question was studiously avoided by that gentleman until an experience had with a case recently sent to him from our Elwyn institution, whereupon I received an ample reply confirming the impression I had formed. A medical officer of the same refuge, within the last few days, examined critically the inmates of the latter institution, and is of the opinion that not a few are moral imbeciles. Dr. Brockway, of the Elmira Reformatory, states that he discovers among his prisoners only six per cent in whom the moral sense is normal. He prefers to speak of many of his cases as moral imbeciles. It is true that we are using this term for the want of something better; but it certainly defines to the common

understanding a class of individuals in whom there is a lack of self-control, a weak volition, and failure in, if not an absence of, the higher elements of a full cerebral development for which we know no more suitable appellation than moral imbecility.

Adopting these views, their practical application soon comes in a sequestration of many whose liberty now is only a vicious license. The tendency at our own institution is toward life detention of all such cases. We refuse for them the ordinary routine of education, because we believe that in educating moral imbecility after the current notions of education, we are training experts who may afterwards figure in the rôle of so-called moral insanity; that by giving such subjects any considerable *school* education we are only arming them for more serious exhibitions of evil. It is a radical, often inherited condition, just as incurable as inherited forms of idiocy or intellectual imbecility. When we shall arrive at this definite conclusion about cranks, habitual drunkards, and the like, and fully apply it, and when we introduce the indeterminate sentence for crime, such sequestration of the morally and socially unfit will be brought about that our human stock will be improved by keeping out of the national blood some terribly bad strains.

It is a noteworthy fact that in most, if not all, the cases which we have determined to be moral imbeciles, there is a neurotic history in the family antecedents; epilepsy, monomanias, inebriety, crime, are frequently the correlative exhibitions in families from which proceed these children of keen intellectual precocities, but inveterate tendencies to vagrancy, prostitution, and lawlessness.

DR. JAMES HENDRIE LLOYD.—With reference to inventing another name for moral insanity, I would say that I do not believe that the condition exists, and therefore I cannot name it. In the last case described by Dr. Kerlin I believe that there is something more than a moral defect. There is something wrong with the intellect. The term moral insanity I consider a misnomer. Guiteau was said to be the subject of moral insanity. The name that I should apply to him would be criminal. As I did not hear the paper, I do not wish to be considered as criticising it. I refer to the subject in general, especially as it concerns the insane and those who are claimed to be insane.

DR. CHARLES K. MILLS.—Dr. Kerlin in his remarks very clearly brought out the strong points in this question; but I believe that Dr. Lloyd's remarks show that he is astray as to the

origin of the facts which some of us have been studying. There may be some who believe in this moral imbecility or moral insanity from the standpoint of the pseudo psychologist, but I do not know these false theorists. The point that I make is this: that Dr. Kerlin, who for twenty-five years or more has been among idiots and imbeciles, and who has studied them in the concrete—who has seen scores or hundreds where others have seen units—has come to his conclusions from a different standpoint. Another point in Dr. Kerlin's remarks was in regard to the question of control. Perhaps the very best legal criteria of responsibility that have ever been given are those formulated by Sir Fitz-James Stephens. Among these, certain have reference to the ability to distinguish between right and wrong, not only in the abstract, but in the particular instance; and a higher criterion, which is gradually coming to be included by others, namely, that a person who does a homicidal or criminal act has lost the power of controlling his actions. I believe that some of his acts are just as much beyond the control of the moral imbecile as are the acts of the hallucinatory lunatic who cannot resist doing that to which he is bidden by a hidden voice. I believe that any one who studies closely many of these cases will come to the same conclusion.

The next paper, by W. A. WILMARTH, M.D., Assistant Superintendent of the Pennsylvania Institution for Feeble-Minded Children, was on

#### THE PATHOLOGY OF IDIOCY.

The following is an abstract of the paper:

The literature of idiocy consists of scattered reports of post-mortem examinations in institution reports and incidental notes in treatises on disease of the nervous system. I have drawn the material for this evening's paper largely from our own post-mortem records, illustrating the cases presented by photographs and actual specimens of lesions and malformations described, and introducing such facts as I have been able to glean from the above-mentioned sources.

Among the causes which may produce mental defect may be mentioned destruction or non-development of a considerable portion of the cerebral cortex; absence or destruction of important commissural tracts; destruction of the organs of special sense or of their ingoing tracts, preventing the reception of impressions

from without; disease of the cerebral vessels, with consequent interference with the nutrition of the ganglionic cells; so necessary for their proper functional activity; atrophy and hypertrophy of the brain, and pressure from cerebral effusion.

#### ANALYSIS OF FORTY CASES.

*Anomalies of the Skull.*—Hypertrophy of the skull was seen in five instances, while the bone was unusually thin in three cases. Deformity of the base, especially about those portions of the ethmoid and sphenoid bones which go to make the base of the skull, exists in nearly all congenital cases. This is regarded by Ireland more as a proof of misdirected formative power than bearing any distinct relation to the mental hebetude.

*Adhesion of the Membranes*, with or without apparent thickening, and varying in extent from a very small localized area to a complete adhesion, exists in forty per cent of our cases. The post-mortem records at Longenhagen, Hanover (thirteen cases), describe this lesion as existing in 84.6 per cent of cases.

*The average weight* of the forty brains was thirty-seven ounces; of the cerebrum, thirty-one ounces; cerebellum, 4.40 ounces; of the pons and medulla, a little less than one ounce. The weight of the cerebellum bears a relatively high proportion to the weight of the cerebrum, but bears no relation to the mental state of the individual. Whenever early and extensive paralysis had existed, the cerebellum was generally small.

*The frontal lobes* very generally appear small, especially in cases of the congenitally feeble-minded. This is evidenced by the short proportionate length of the lobes measured along their superior border, and by the frequent exposure of the insula by the imperfect development of its lower convolution.

*Defect of the commissural system* is common. In the corpus callosum it was found in three instances; in the middle commissure in four cases. The posterior and optic commissures were absent in one case.

*Irregular arrangement of the fissures and convolutions*, consisting of a decided departure from their usual plan, is occasionally seen; confluence of the principal fissures, and more frequently still of the secondary fissures, is common. In about ten per cent of our forty cases a strong tendency of the fissures to assume a vertical position is shown; the Sylvian or superior temporal convolution may be prolonged to the superior border of the hemisphere, or

long, deep fissures may be found in unusual places, where all the fissures whose natural direction is vertical become strongly marked. All these appearances are most marked in children of a low grade of intelligence, and may probably be regarded as an evidence of misdirected formative growth. In two cases of "Mongolian" type of imbeciles the cerebrum in each case was found well developed in its relative proportion, the cerebellum smaller than usual, while the pons and medulla reached only about half the usual weight. No microscopic examination has as yet been made.

*General atrophy* was found in one case, and internal hydrocephalus with flattening of the convolutions from internal pressure in one instance.

Among the *microscopic appearances* found in idiocy were relative sparseness of ganglionic cells, disproportionate thickness of the outer barren layer, and the presence of nuclear bodies surrounded by a little granular protoplasm, and contained in clear, round spaces in the neuroglia, described by Spitzka. Degenerated and abortive ganglionic nerve-cells, increased density of the neuroglia with abundance of small round cells, thickening of the walls of the blood-vessels, which are sometimes dilated in places, again contracted or occluded. Sclerosis of all degrees of progress. Atrophy of nerve-fibres of the white substance. In at least three cases such destruction of the ganglionic cells in portions of the cortical substance, with condensation of the connective tissue, and in one instance, deposit of crystals from absorption of effused blood, that it seems doubtful if any functional activity remained in the affected parts.

Specimens were shown illustrating atrophy of convolution from pressure of bony plates in the dura mater, with malformation of occipital lobes; destructive lesion of frontal lobes, with non-development of the insula and other portions of the hemispheres; also a microcephalic brain, with arrested development of convolutions and general atrophy; another, a fairly well-formed brain from an idiot of the lowest grade, where apparent non-development of the cortical substance of portions of both hemispheres existed.

#### DISCUSSION.

DR. FRANCIS X. DERCUM.—This is certainly a remarkable and interesting paper. I notice that in one of the specimens sent around there is a convolution which I have not seen in any of the

brains which I have examined, and this is the inferior internal *pli de passage*. This brings to mind a speculation of my own. It occurred to me, Why should we have a convolution such as the internal *pli de passage*? It is found in monkeys of low grade. Why should we find this convolution recurring in the human brain? It seemed to me that if there be some inherent defect in the power of development of the cerebral vesicles, that defect must make itself most manifest along certain lines. Thus there will be a tendency to absence of certain portions. This defect would be made more evident if the cranium ossified too soon. In such case we should expect the appearance of transverse and perpendicular fissures corresponding to what is found in the brains of monkeys. The question then arises, Why is it that we have ape-like convolutions present? It seems to me that if any one part be suppressed, that the brain being a balanced whole, some other part must be developed, and, therefore, ape-like convolutions are necessitated by ape-like fissures.

DR. CHARLES K. MILLS.—The work at the Pennsylvania Institution for Feeble-Minded Children is of great value, not only for what it is, but for what it will lead to and suggest. I have always said that as much can be done towards settling the proverbs of localization in an asylum for idiots as by experiments on animals or by any form of experimental work, although I do not by any means disregard the value of such experiments. The facts brought out in a general way by this paper show the possibility of our eventually getting at a wider and fuller differentiation of the faculties or so-called faculties of the brain beyond mere motor and sensory localization. As Dr. Wilmarth referred to Dr. Benedikt, it may be interesting to state that about eight months ago I received a letter from Dr. Benedikt, in which he says that he does not advocate the idea that there is an absolutely fixed type of criminal brain, but his observations tend to show that peculiarities of fissures and convolutions occur in low-type brains, and special peculiarities in certain cases.

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Original Articles.

ON THE TREATMENT OF PROGRESSIVE LOCOMOTOR ATAXIA WITH RAREFIED AIR,  
AFTER THE METHOD OF JUNOD.

By HENRY M. LYMAN, A.M., M.D.

CHICAGO, ILL.

IN the issue of *The Medical Record* for September 30th, 1882, pp. 373-4, I reported a case of progressive locomotor ataxia which seemed to have been improved by rest and vigorous application of dry cups. Briefly stated, the history was as follows:

D—, a private soldier in the army, but free from all venereal taint, when about twenty-three years of age, was obliged to lie out one wet night, during the cold spring weather in 1865. This caused a pain in the back, with other symptoms of sufficient severity to drive him for a number of days into the hospital. No definite conclusion relative to the nature of the disorder appeared to have been reached by the medical attendant, and he finally returned to duty, to be discharged from the service soon afterwards at the close of the war.

With the exception of some pain and weakness in the lumbar region, he enjoyed good health for several years, and became actively engaged in mercantile pursuits. He was severely overtaken during the period of confusion following the great Chicago fire, in 1871, and began to suffer with lancinating pains and with paroxysms of pain-

ful micturition which were in no way relieved by active treatment for a supposed spasmodic stricture of the urethra. Notwithstanding these recurring pains, he presented the appearance of excellent health, and continued his usual avocations. During the year 1879, he became alarmed by an increasing unsteadiness in his gait, especially in the dark. I now saw him for the first time, and found present the ordinary phenomena of progressive locomotor ataxia: lightning pains, anæsthetic, paræsthetic, and analgesic patches upon the surface of the limbs, paroxysms of hyperæsthesia affecting the mucous surfaces of the mouth and pharynx, absence of the patellar reflex, ataxic gait, diminished power of co-ordinating the muscles of the upper extremities, cracking of the joints, unequally contracted pupils, with loss of the pupillary reflex, progressive atrophy of the optic discs, and failure of sexual appetite.

Treatment with galvanism, nitrate of silver, iodide of potassium, and bichloride of mercury did no good. The patient continued actively engaged in his business, travelling from California to New York and elsewhere; and finally, in October, 1880, was married, though compelled to keep his bed, without the slightest desire or ability to consummate the marriage.

About the same time, Mr. D—— found himself suddenly deprived of the means of support by the failure of the mercantile house with which he had been connected. The effect of this disaster was decisive. "Eyesight began to fail, ataxia increased, the muscles of the lower extremities began to dwindle, lightning pains were frequent and agonizing. The rectum became anæsthetic and uncertain in its retention of fæces. The feet were cold and numb." Walking was almost impossible.

While in this condition, D—— was persuaded to make trial of Junod's boot, applied daily to the lower extremities, along with vigorous dry-cupping of the spine. This mode of treatment produced a gradual improvement in the condition of the patient, so that, when he again came under my observation, during the summer of 1882, I was able to record his condition, as follows:



"There is still some degree of ataxia manifested in the movement of the feet, especially when excited or weary. The upper extremities are perfectly manageable. Anæsthesia has disappeared from the regions where it was formerly manifested, but the tendinous reflexes are still absent. The patient walks easily and rapidly without a cane, turning sharply around without difficulty, but he is unsteady on his feet with his eyes shut, and he is unwilling to trust himself in the dark. The eyeballs move perfectly, but the pupils are contracted, and do not respond readily to changes of light, though they move somewhat in accommodation. There is considerable atrophy of the optic discs. Difficulty is apparent in recalling proper names, but otherwise the intellectual processes are intact. Appetite, digestion, and sleep are perfectly natural. The patient no longer feels that excessive sensibility to changes of temperature which formerly troubled him. Erections take place as often and as completely as ever. Amatory feelings have revived, and sexual intercourse has been performed several times without much subsequent lassitude, but for prudential reasons has been discontinued. The patient feels competent to resume business, but is wise enough to see that his disease is not cured. He still continues the use of the cupping apparatus every other day, and is positive in his conviction that his improvement, though gradual, is progressive."

Thus far the course of the disease at the date of my report in 1882. Since then, I have had opportunities, at intervals of a few months, for noting its further evolution. During the last five years, there has been a complete development of the second stage of the disease, and a partial entrance upon its final, paralytic stage. The patient has lived in various parts of the country, and has taken very little medicine, but has made constant use of the cupping apparatus with which he had furnished himself six years ago. He has usually applied the boots to the lower limbs as often as every other day. It is to the effect of this treatment that I now desire to call especial attention.

So far as the onward march of the spinal disease is concerned, the treatment has produced no appreciable result. This is true of this case as it is, also, of every other case of genuine tabes dorsalis in which I have seen it tried. If employed during the first and second stages of the disease, when the characteristic fulgurant pains form a prominent feature, great relief is sometimes experienced, but no other immediate result can be discovered. It is important in this connection to avoid the error of mistaking the natural subsidence of pain in advanced stages of the disease for the effect of treatment. But, when the paralytic stadium is imminent, my observation of this patient leads me to believe that considerable benefit may be derived from treatment of the limbs with rarefied air. When this treatment was commenced, six years ago, the muscles of the lower limbs of my patient were becoming incapable of supporting the weight of the body, and the circulation of blood in the extremities was insufficient to maintain their natural color and temperature. Daily application of the boots, with rarefied air, produced a gradual improvement until the patient was again able to stand, and to walk as well as before the collapse of his limbs. At the present time he is able to walk, though with the usual ataxic gait, and the muscles of the legs are firm and solid. Above the knees the muscles of the thighs present the same condition as high as the middle of the upper third of the thigh—that is, as high as the boots can reach when applied to the limbs. Above this point, the abrupt transition in the continuity of the muscles from a full and natural volume to the thin and flaccid structure of atrophy is very remarkable. The muscles of the hips exhibit the same progressive atrophy, and it is probably invading the muscles of the trunk. Only those regions which have been subjected to the action of rarefied air have escaped; and the line of demarcation is too well defined to admit of any doubt in the premises.

The question that now arises has reference to the manner in which the use of the cupping apparatus operates to hinder muscular atrophy. Obviously this is accomplished

by a reinforcement of the processes of circulation. Is this result brought about indirectly, through reflex influences, or is it a direct effect of local changes consequent upon dilatation of the muscular vessels during the application of the boots? Probably, it follows both of these methods of modifying nutrition. The value of counter-irritation applied to the lower extremities in cases of locomotor ataxia has been lately emphasized by M. Brown-Séquard, in his recent remarks before the Society of Biology, in Paris. He is reported to have said (*Lancet*, June 4th, 1887, p. 1,161) "that it was important to apply revulsive treatment, not to the back, as was generally done, but to the lower extremities. The cord is acted upon in this way by all the sensory nerves, and the effect greatly increased. He quoted the case of a medical man, who was cured of locomotor ataxy by circular blisters around the leg and thigh." But, in the experience of my patient, it is evident that the general effect of counter-irritation has been insufficient to prevent atrophy in those parts of the limbs which had not been included within the receiver of the cupping apparatus, though its direct effect upon the included portions has been very beneficial. It is not improbable that the cases in which revulsives are most productive of good will prove to be examples of peripheral neuritis, in which it is well known that reflex influences produce a most salutary effect.

It is hardly possible, at present, to give a thoroughly satisfactory explanation of the manner in which rarefied air acts upon a given portion of the body, to improve its nutrition. When the whole person is immersed in highly rarefied air, the results are very deleterious (Landois and Stirling's *Physiology*, 2d ed., p. 229). How, then, can a partial immersion become useful? We learn from the experiments of Bernard ("Dic. Encyc. Sci. Med.," Art. *Musculaire*, p. 662) and others, that, if the nerve leading to a muscle be divided, the blood that returns through the muscular vein exhibits less than the ordinary venous appearance; it has not surrendered its full quota of oxygen in exchange for carbon dioxide, etc., generated in the

muscle. The same thing, only proceeding after a more tardy fashion, occurs when the anterior nerve roots in the spinal cord are invaded by disease. Oxygen starvation supervenes, and this produces a granulo-fatty degeneration; hence, the characteristic muscular atrophy and paralysis that mark the later stage of progressive locomotor ataxia. Electricity, massage, cupping, and other similar expedients serve to replace the nervous influences that are lacking, and are thus useful in delaying the advance of muscular atrophy. But the experience of my patient indicates that something more than mere increase of the circulation is needful to secure the adequate nutrition of the muscular fibre. Those muscles and portions of muscles which were left to the action of electricity and manipulation have become atrophied in the usual way; while the muscles that have been treated with rarefied air have retained a great share of their normal firmness and functional vigor. There must, therefore, be a local process set up through rarefaction of the air surrounding a muscle, to which process the conservation of nutrition must be ascribed. This local change implies something more than mere acceleration of the blood current—in fact, it is doubtful whether the circulation is materially quickened by the act of cupping. Blood is drawn into the limb, but it is also detained in the part. It is probable that the nerve-endings in the muscles may be considerably stimulated by this operation; but it is not easy to understand how this can be any more beneficial than excitation with electricity or by manipulation. If a diminution of atmospheric pressure over a portion of the body exerts the same effect upon the interchange of elements in its tissues that is observed when the whole body is thus relieved from pressure, it is impossible to infer that the good results of the operation are due to an improved respiration of the tissues; for, when the entire surface of the body is subjected to the action of rarefied air (Landois and Stirling's *Physiology*, loc. cit.), the oxygen in the blood is diminished, carbon dioxide is imperfectly removed, and oxidation within the body is lessened. It, therefore, seems probable that when

the limbs alone are subjected to a reduction of atmospheric pressure while the trunk remains free, the conditions are sufficiently different to account for the observed fact of improved nutrition. Under such conditions the blood is sucked out of the deeper tissues of the limb, and is forced out of the member, where it cannot be effectually aërated, into the general circulation of the body, where aëration proceeds under normal conditions. The nutrition of muscles which have been thus artificially defecated must be favorably affected by such treatment.

But, after all, this leaves still unexplained the fact that such improvement is confined to the parts that have been directly exposed to the action of rarefied air. We are, therefore, compelled to conclude that the process consists in an artificial dilatation of the muscular fibre, thereby facilitating that intussusception of nutriment through which the bulk and vigor of the muscle are sustained.

From these considerations we may conclude:

I. That progressive locomotor ataxia cannot be cured by dry cupping.

II. That the painful sensations which accompany the evolution of the disease may sometimes be relieved by this method of treatment.

III. That the principal advantage resulting from the use of Junod's cupping apparatus consists in the improved nutrition of the muscles of the limbs which are subjected to its action.

IV. That the delay of muscular atrophy thus procured is due to the local action of rarefied air upon all the structures of the affected parts, and is limited to the tissues which are actually inclosed within the exhausted receiver.

V. That the improvement of nutrition is, therefore, principally accomplished by direct action, rather than by reflex influences exerted through the spinal cord.

VI. That in the use of rarefied air we employ an agent which is competent to fill a subordinate and limited, yet often serviceable position in the treatment of cases that are characterized by a tendency to muscular degeneration.

This, probably, is also true in other diseases besides progressive locomotor ataxia.

## PARAMYOCLONUS MULTIPLEX, WITH A REPORT OF A CASE

BY M. ALLEN STARR, M.D., PH.D.,

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM, NEW YORK POLYCLINIC.

**P**ARAMYOCLONUS multiplex is a spasmodic affection of the muscular system of peculiar character, distribution, and course, dependent upon irritation of the nervous motor mechanisms.

The chief features of this disease are illustrated by the following case:

John D., aged 33, of Kingston, Canada, a grocer, of good family history, but of nervous temperament, was in his usual health until September 15th, 1886, when he strained his back and right shoulder by lifting a box, sixty pounds in weight, while in an awkward position. The pain, below the right shoulder blade, resulting from this strain was so severe that he went at once to his physician, Dr. H. J. Saunders, in whose office he had a peculiar attack. He began to cry and scream with pain and soon felt a choking sensation and was unable to get his breath; then followed convulsive movements of the body and legs, the latter being drawn up and thrust out forcibly. These lasted an hour, after which he was taken home. They returned at short intervals, for three days, being attended by pain in the back; and then they began to involve the muscles of the upper extremities and neck; and a few twitching movements of the face also occurred. The spasms were always very rapid, but were chiefly confined to muscles attached to the trunk, it being noticed from the first that the muscles of the forearms and hands and of the legs and feet did not take part in the spasms. The diaphragm was affected usually, so that dyspnœa and

exhaustion attended the attack, and it was occasionally followed by vomiting, when occurring after a meal. He had at first ten or twelve such attacks in twenty-four hours, each lasting nearly an hour. After two or three weeks, the attacks became less severe, the legs being less violently moved, and their duration decreased. But they had continued until May 2d, 1887, when I saw him, varying in severity and duration, but constantly diminishing, so that now he may have but three or four a day, though sometimes they are more frequent. He had several attacks in my presence, each lasting from one to five minutes, and leaving him much exhausted. The muscles first affected were those of the back and abdomen, a series of quick, alternate contractions of the dorsal muscles and of the recti abdominis, resulting in a rapid protrusion and retraction of the abdomen. As this became more rapid and forcible, the body and head were thrown backward and forward, without any spasm of the muscles of the neck, and in one attack which occurred while he was standing, similar complementary movements to preserve his balance produced alternate slight flexion and extension of the hips. In a much more severe attack which was observed when he was stripped, the muscles involved were first those of the back and abdomen already mentioned; then of the neck, so that the head was not only nodded, but was turned from side to side; then of the muscles of the upper arm, the pectorals, deltoid, biceps, and triceps, being all in action, and lastly, the muscles of the thigh, the quadriceps femoris, biceps, and semi-tendinosus and semi-membranosus, with the glutei acting with such force as to cause movements of both hip and knee joints. The contractions of these muscles were rapid, the rate rising to ninety per minute. The spasm in the arms was not severe enough to move the shoulder or elbow joints, but the muscles named were seen to contract and raise the skin. He said that the wrist and fingers, ankles and toes, had never participated in the spasm. He had noticed that formerly there was some movement on the forearms, probably of the supinator longus muscles, but this I did not see.

There was also, in a severe attack, a spasm of the diaphragm resulting in a long inspiration, accompanied by a sound, and while this was tonic, rapid movements of the intercostal and accessory respiratory muscles to supply the lack of inspiratory action of the diaphragm were made. Only at the very first has there been seen any spasm of the facial muscles, and for some months these have not been affected.

The spasms were so severe as to make me fear that he would fall while standing, as he said he had done several times, and would nearly throw him out of an arm chair when sitting. They come on suddenly, but are usually preceded by a peculiar sensation which ascends from the legs to the head and ceases as suddenly, leaving him in a state of considerable exhaustion, panting and perspiring, and looking badly as if about to faint. During the interval between the spasms an occasional fibrillary twitching in the muscles of the back and pectorals was observed, but not elsewhere. He could not stop the spasm voluntarily or in any way limit its course. Nor could he start it voluntarily. But any exposure of the skin to cold, any irritation of the skin by electricity, any tapping of the tendons at the knee or attempt to elicit ankle clonus was sufficient to start a spasm at once. He said it often came on after muscular effort, such as a long walk. It was also more likely to occur after mental excitement, and it usually came on when he began to talk about his condition or went to see a stranger. From the beginning until the present time, pressure on the right shoulder blade, where he had the pain, produced a spasm at once. It ceased during his sleep and never woke him by occurring at night. He thought that a drink of whiskey arrested it sometimes.

His motor power, sensory perception, and voluntary co-ordination were not in any way impaired. His skin and tendon reflexes were exaggerated, a marked ankle clonus being present on both sides. His muscles responded normally to both electrical currents. Mentally, he was perfectly clear, but somewhat excitable, and after



each attack in his exhausted condition, tears came to his eyes as he spoke of his trouble. He did not appear, however, to be of an hysterical temperament. Treatment had been somewhat successful, as he considered his condition much better than it had been six months ago, for he said that the attacks were less severe, shorter, and less frequent.

This spasmodic affection, limited to the muscles of the body and proximal portion of the limbs and only occasionally affecting the neck and face, has been termed by Friedreich,<sup>1</sup> who described it first as paramyoclonus multiplex. Since its first description, only seven cases have been reported, those of Lowenfeld,<sup>2</sup> Marie,<sup>3</sup> Silvestrini,<sup>4</sup> Bechterew,<sup>5</sup> Seeligmuller,<sup>6</sup> Homen,<sup>7</sup> and Remak.<sup>8</sup> My own case brings the total number up to nine.\* It must be regarded as a distinct disease, since it is widely different from chorea, from hysterical spasms, from epileptiform convulsions, from convulsive tremor or tetanilla, and from tic convulsif.

Its causation is uncertain. In three cases fright, in one case severe hemorrhage which may have caused fright, in one case the shock of a cold bath, and in my own case an overstrain causing pain have preceded the development of the spasm. In the other three cases, no cause could be found. In two of the cases, a chronic spasmodic affection had been present for several years. It has been observed but once in a female, the remaining eight cases being males. The age of the individual has little to do with its development, for persons of all ages from ten to fifty-two have been affected. Its symptoms are quite characteristic. The spasms are bilateral and symmetrical. They are limited to certain muscles. In eight cases, the quadriceps femoris and flexors of the leg, and the so-called "upper arm group of muscles," the deltoid, biceps, and supinator longus, were affected. In seven cases, the muscles of the back were involved. In six cases, the muscles of the neck contracted. In five cases, the glutei. In

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\* The case of Silvestrini is so imperfectly described in abstracts as to be useless for comparison, and the original was not accessible to me.

four cases, the face and the diaphragm were involved. In no case have the muscles of the hands or forearms, of the feet or legs been affected. The usual limitation of the spasm to the body muscles with those of the thighs and arms is very noticeable.

The character of the spasm is also characteristic. It is a rapidly repeated clonic spasm occurring at intervals. In six cases, the rate of contraction has been counted. It has varied from 50 per minute to 180 per minute. In my own case, it was about 90. It is not a sudden, single irregular muscular contraction, like that of chorea, but appears to be always bilateral and to involve several muscles of a physiological group at once, thus resulting in a series of movements, any one of which can be voluntarily made. In several cases, a tonic contraction has occurred in one or more of the muscles affected, before or during the clonic seizure. In my own case, the spasm of the diaphragm was tonic for one-eighth to one-quarter of a minute during each attack, and in the early attacks, the spasms of the back were tonic for some seconds. The clonic contractions continue, when once set up, for a varying time, from half a minute to ten minutes, and are succeeded by a complete interval of freedom from spasm. In my own case, this interval had varied from half an hour to about one week. And the fact that the free quiet intervals were getting longer had encouraged him to hope for a recovery. During the spasm itself, the resulting movements were of a very violent nature. The head was thrown about by the movements of the body, rendering the patient dizzy. The body was tossed about in the chair, so that there seemed to be danger of his being thrown out upon the floor. If the spasm occurred while he was walking, he was quite liable to be thrown down, and had hurt himself several times. But this violence is not always present—for in two cases, the spasms were never severe enough to cause a movement of the joints, and were only observed when the patient was stripped—being then of the nature of a fascicular twitching. In my own case, such a fascicular muscular twitch-

ing was occasionally seen during the intervals in the muscles of the back and the pectorals.

In the majority of the cases, any tapping of the tendons or any irritation of the skin was sufficient to produce a spasm. This seems to be an important point, for I am not aware that it has been observed in hysterical or choreic spasms. It is true that, in hysterical cases, certain zones or areas can occasionally be found on the body, irritation of which may cause or may arrest the attack. But in this condition the spasm is produced by irritation anywhere on the skin—or by tapping the tendons at the knee and ankle—and was not associated with disturbances of sensation, which are characteristic of hysterical zones. The knee jerk has been increased in four cases, was less in one case, and was not tested in the remainder. The skin reflexes were also increased in four cases, and are mentioned as normal in but one of the remainder. Mental excitement seems to have predisposed to the onset of the spasm in three cases. Had the disease been hysterical in nature, this would probably have been observed in a larger proportion. Voluntary effort stopped the spasm in four cases and made it worse in three cases. Had the disease been hysterical, volition would probably not have influenced it favorably in the majority of cases. The spasm has ceased during sleep in four cases, but has continued in one case.

In none of the cases have consciousness, motion, sensation, co-ordination, or electric excitability been in any way affected—an important negative fact, since it proves at once that the condition is a functional neurosis, and makes it very unlikely that it is of an epileptic or an hysterical nature. In one case, which died of phthisis, a careful examination by Prof. Schultze, of Heidelberg, failed to reveal any lesion of the nervous system.

It is evident from this review of the symptomatology that the characteristics of the disease are quite distinct; that it can be differentiated from chorea, from hysteria, and from epilepsy. Is there any disease known which it at all simulates? In tic convulsif, we have an affection of the

face consisting of spasmodic contractions of irregular intensity and frequency, often attended by intervals of freedom. The resemblance to paramyoclonus multiplex is more than superficial, and has been noticed by several writers. But all seem to agree that in tic convulsif the face is usually chiefly, if not exclusively, affected; that the contractions are often single and unilateral, are liable to occur during voluntary motion; that their intensity is not varied, but is quite uniform; that the spasm is not produced or increased by external influences; and that it is always a co-ordinated volitional motion which is produced. Guinon,\* it is true, has described a *maladie des tics convulsifs*, in which title he wishes to include those spasmodic affections described by various authors as jumping, coprolalia, myriachit. But here again there is a wide difference from paramyoclonus multiplex. It seems, therefore, as though the disease must be regarded as distinct from tic convulsif, and as having a character of its own.

The question has arisen whether it is identical with an affection described by Hammond in 1867 as convulsive tremor. This was suggested by Dr. C. L. Dana, who saw my case and identified it as paramyoclonus multiplex. I have carefully studied the various cases described under this head by Hammond in his original article<sup>9</sup> and in the last edition of his work on "Nervous Diseases,"<sup>10</sup> and in a recent report of a case.<sup>11</sup> These cases are by no means uniform, and do not seem to me to belong to a single class of disease. There are but three which in any way resemble the disease under discussion. In two of these (cases V. and VI.), there was no limitation of the spasms to the muscles affected in paramyoclonus—the arms and legs being affected as well as other muscles; there were motor and sensory symptoms, which were well marked, in addition to the spasms; and there were cerebral symptoms of such intensity as to lead the author originally to ascribe the disease to the cerebellum, a position which, however, he has retracted at present. It seems to me, therefore, that Hammond's original cases were either not paramyo-

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\* Rev. de Méd., 1886, January.

clonus multiplex or, if they were, no one from his description of them could arrive at the characteristic features of this disease. It is, therefore, necessary—much as we might prefer to claim for our own country the original observations—to admit that Friedreich was the first to describe the affection, and to adopt the name which he chose.

A case reported by Hammond only last year,<sup>11</sup> under the title of convulsive tremor, does, however, seem to conform to the picture of this disease. "A female, aged 22, was affected by an involuntary contraction of the gluteal and spinal muscles, by which the body was violently raised from the recumbent or sitting posture and, at the same time, drawn violently backward, and on the completion of this movement, the body, with equal violence and suddenness, was bent forward by the contraction of the abdominal and pelvic muscles, and then a period of rest of variable duration, sometimes being only a few seconds, and others several minutes, intervened. There were no other disturbances of mind or body, and no hysterical manifestations. Attacks were increased by mental excitement, and at a minimum when the mind was calm. Muscular activity did not influence them. They were absent during sleep." They could be controlled by the will only for a short time, the effort producing a feeling of nervousness. No cause was found for the condition which, after resisting bromides, yielded to hypodermatic injections of arsenic.

Here the picture is sufficiently distinct and conforms quite closely to that of paramyoclonus multiplex, while it differs widely from that of convulsive tremor, as described in the author's text-book. If this be accepted as a case of paramyoclonus multiplex, it brings the number of cases up to ten.

The only other cases of convulsive tremor on record are two, which were reported from Dr. Hammond's clinique by his assistant Dr. Brown;<sup>12</sup> but neither of them are cases of paramyoclonus multiplex. In one of them, a manifestly hysterical case, the spasm was limited to one arm. The other case is one of chorea electrica, first described

by Henoch. And as this disease might be mistaken for paramyoclonus multiplex by a superficial observer, it may be well to mention its characteristics. Henoch says<sup>19</sup> that in chorea electrica we have a combination of true choreic movements with clonic twitchings. The patients are quiet, and lightning-like twitches occur from time to time, perhaps every five minutes or more frequently, especially on the muscles of the neck and shoulders. There is also seen a twitching of individual muscles when the body is naked, not sufficient to move the limbs. These continue during sleep. The disease occurs between the ages of nine and fifteen, and is a manifestation of direct or reflex irritation of the nervous centres. To this description Dr. Brown's case corresponds quite closely, there being associated with a severe condition of chorea attacks of tonic, followed by clonic spasms, while in the interval fibrillary tremors were constantly observed. The spasms affected the head, hands, and feet, and implicated all the voluntary muscles, including the diaphragm and larynx, so that the patient would growl and bark. The tendon reflex was "slight," and there was "some anæsthesia," the location of which is not stated. The boy recovered under arsenical treatment.

It is evident that a number of different conditions have been brought together under the term convulsive tremor, and of the cases described I find but one in which the limitation of the spasm to certain muscles and the character of the attacks enables one to identify the disease as paramyoclonus multiplex.

But one other affection requires notice in connection with this disease. In a paper, read on Oct. 18th, 1886, before the Medical Society of the County of New York, Dr. Julius Althaus, of London, described an affection which he named tetanilla. He said that it was the same as paramyoclonus multiplex; that he had never published any observations on the subject up to that time, but that he had privately given the name tetanilla to the affection as more euphonious than that selected by Friedreich. He claimed to have seen a number of cases, and of these he alluded in a very cursory manner to five. Of these cases,

three were cases of unilateral spasm, and as one of the characteristics of paramyoclonus is the bilateral symmetrical nature of the convulsion, these cases may be ruled out at once. One of the remaining two cases is not sufficiently detailed to warrant any conclusion as to its nature. The last case (the first in his paper) cannot be considered one of the disease now under discussion. For Dr. Althaus says that all the muscles of the body were affected either simultaneously or successively, and the spasms commonly began in the muscles of the thumbs, each contraction lasting five or six seconds, and being succeeded by six or seven more contractions. Here there is no limitation of the spasms to the trunk muscles, no record of a series of rapid clonic spasms succeeded by a period of repose, no evidence of the presence of fibrillary tremors in the interval. It is evident that Dr. Althaus is mistaken in supposing that his cases were paramyoclonus multiplex. And the remark made by Dr. Hammond during the discussion, that he had not seen any such cases, leads to the conclusion that they were probably not of the nature of convulsive tremor.

It is evident from this review that the characteristic features of paramyoclonus multiplex have not been generally appreciated, and hence it has been mistaken for other affections, and diseases widely different from it, and only resembling it in respect to the common symptom of clonic spasms, have been classed with it. It may perhaps seem impossible from ten cases to form a picture of the disease, but doubtless these cases will be added to and the features of the affection more carefully outlined when its chief characteristics are more definitely grasped.

These characteristics may be summed up as follows: Paramyoclonus multiplex is a spasmodic affection of the muscular system, occurring bilaterally in symmetrically situated muscles attached at one or both ends to the trunk, and in muscles whose function is associated with these, consisting of a series of violent clonic spasms of considerable rapidity and severity, occurring only at intervals; and associated with fascicular tremors of the affected

muscles, persisting during the interval between the spasms. It occurs after some mental or physical strain, and is not accompanied by any disturbance of sensory or motor functions, excepting by an increase of the superficial and deep reflexes. It can be excited by irritation of the skin or tendons.

In regard to the prognosis, it may be said that this is favorable. The majority of the cases have recovered quite rapidly under treatment. In two cases, however, relapses have occurred.

The treatment which has been of most service has been the application of strong galvanic currents to the spine and neck, and the application of the anode to sensitive points in case these exist. Many nerve sedatives have been used, and also nerve tonics. The exact effect of these seems to be doubtful. In my own case, sedatives, tonics, and electrical applications had all been equally futile to arrest the attacks, but the patient had improved to a considerable degree under the varied treatment. I prescribed galvanism to the spine, arsenic and chloral with some beneficial effect. The hypodermatic use of arsenic under the conditions described by Hammond deserves a trial.

It is useless to discuss the nature of the disease from so few cases as are at our disposal. It has been regarded as a functional neurosis, and to this all must agree, both on account of the absence of any lesion, in one case examined by the most competent neuro-pathologist in Germany, and on account of the absence of symptoms of organic disease and the recovery of the cases. Whether it has a central origin and is produced by a hyper-excitability of the brain or spinal cord, induced by the sudden vaso-motor spasm accompanying fright or mental or physical strain, as Friedreich believed, or whether it may be a reflex spasm due to some peripheral irritation which, being conveyed to the spinal and medullary centres, produces the spasm reflexly, as another author has suggested, remains for the future to decide. The case here reported would seem to favor the latter view.



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## THE THERMO-INHIBITORY APPARATUS.

BY ISAAC OTT, M.D., AND CHARLES COLLMAR, M.D.

IT was first observed by Sir Benjamin Brodie in 1837, after an accidental injury to the spinal cord of a patient, that the temperature between the scrotum and the leg was  $111^{\circ}$  F. The breathing in this case was carried on by the diaphragm, and he lived twenty-two hours after the injury. Towards death his respirations were 5 to 6 per minute. Prof. Dunglison has seen in many hemiplegics an increased temperature on the paralyzed side. It has been noted by a large number of observers that if an animal has its spinal cord divided, the temperature of the ambient air being high, that the rectal temperature rose. If the air temperature was low, the animal's temperature fell. It has been shown by Naunyn and Quincke that the rise is mainly due to an action going on in the animal and not to the temperature of the air being higher than usual. In this paper we propose to give the results of partial division of the spinal cord, or rather, section of its different columns, and its effect upon the rectal temperature, heat production, and heat dissipation. One of us, Dr. Ott, has already shown that section of the lateral columns of the cord was followed usually by increased exhalation of carbonic acid. This increase of carbonic acid was not due to circulatory disturbance, for the blood pressure after partial division of cord falls, which would not favor increased changes in the tissues and consequent increase of carbonic anhydride. The blood runs rapidly through the widened channels, for the vaso-motor nerves in the lateral columns have been either injured or divided, and it is not probable that tissue-change is increased. Neither does the activity of the respiratory apparatus cause the increase

of carbonic acid, as the section diminishes the number and amplitude of the chest movements.<sup>9</sup> These data show that the increased amount of carbonic acid expired is due to the removal of some influence which restrains the chemical movements of the tissues.

*Method.*—Etherized cats and rabbits were used, and the number of experiments was twenty-five. The animals were operated upon in a room eight feet wide and thirty-two feet long, with a free circulation of air. A thermometer placed immediately above the operating table noted the temperature in which the animal was during the period of observation. To determine how much the temperature of an animal rose, we opened the spinal canal of an animal, leaving the dura mater intact, and observed that the temperature of the rectum rose a degree in an air temperature of 102 for an hour. It is difficult to accurately estimate the increase, as the animal's struggles will also increase it. In all the animals the cord was divided at about the eighth dorsal vertebra. When the spinal gray was divided, the rise of temperature was .8° F. in Exp. 6, and a fall of 2.8° F. in Exp. 3.

When the anterior columns were nearly divided, there was a rise of .9° F., Exp. 12, whilst when the posterior columns were divided there was a fall of .3° F., Exp. 11. When the lateral columns above or in conjunction with the gray or part of the posterior columns were divided, then the rises of temperature were .7° F. in Exp. 1, 1.2° F. in Exp. 9, 1° F. in Exp. 4, 2.9° F. in Exp. 5, 2° F. in Exp. 2, and 2.4° F. in Exp. 7. All these experiments and others exhibited the greatest rise when the lateral columns were divided. Hence it is fair to infer that in the lateral columns run fibres whose division permit a rise of temperature behind the point of section. The question now arises, To what is this increment of temperature due? Is it generated by increased production or diminished dissipation? To determine this, we used d'Arsonval's calorimeter surrounded by felt, feathers, and saw dust. The air was aspirated through a narrow leaden tube coiled around the chamber containing the animal, and immersed

in the water chamber. By means of a trap door in the roof the temperature of the room was kept about a degree above that of the calorimeter. The error of this instrument for each degree that the air is above that of the calorimeter is  $.025^{\circ}$  F., which cannot affect the accuracy of our results. The tabulated results are as follows :

Exp. 1, Cat, weight, 3.32 lbs.

First hour.		
A. T.	R. T.	C. T.
101.4	102.5	99.5
101.	102.1	99.8
Heat dissipation,		12.51
Heat production,		19.67

Lateral columns divided.

101.4	101.6	99.875
100.7	104.7	100.15
Heat dissipation,		11.47
Heat production,		20.04

It will be noted here that the temperature of the animal normally rose, because there is not a free circulation of air in the calorimeter, although quite sufficient for the purposes of respiration.

Exp. 2, Cat, weight, 4 lbs.

A. T.	C. T.	R. T.
100.	99.	102.2
100.7	99.5	105.5
Heat dissipation,		22.94
Heat production,		32.90

Lateral columns partially divided.

100.5	99.55	102.8
100.7	100.2	106.8
Heat dissipation,		27.11
Heat production,		40.39

Exp. 3, Cat.

A. T.	C. T.	R. T.
99.	98.2	103.8
101.	99.2	105.5
Heat dissipation,		8.34
Heat production,		14.68

Lateral columns partially divided.

101.	98.7	103.2
101.3	99.2	105.6
Heat dissipation,		20.86
Heat production,		31.66

Exp. 4, Cat, weight, 3.44

A. T.	C. T.	R. T.
101.5	98.575	100.2
100.3	99.	105.4
Heat dissipation,		17.73
Heat production,		32.57

Lateral columns partially divided.

101.	99.	103.4
100.8	99.65	107.8
Heat dissipation,		27.11
Heat production;		39.67

The increase and decrease of heat units are expressed by curves in Fig. 1, the dotted line being that of heat dissipation, the other line being that of heat production. By examining the curves, we see that both heat dissipation and heat production are increased, the latter more than the former. The temperature curve is given below.

The question follows, How is this increased temperature due to increased production produced? I have already shown that the increase of carbonic anhydride cannot be due to vaso-motor changes or those of a respiratory nature, and for the same reasons are not the origin of the increased production of heat. Prof. R. Meade Smith,<sup>1</sup> in a series of researches, reached the conclusion that with a larger supply of blood the cool skin, even though exposed to excessive and rapid loss of heat, will become warmer, while, on the other hand, the warmer muscle will become cooler. Consequently, he states the conception must be erroneous which is generally held as to the temperature changes in muscle from alterations in the blood supply after section of the nerves. We believe that there is every reason to assume heat centres to exist in the spinal cord which are connected with the production of heat, and sec-

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<sup>1</sup> Archives of Medicine, 1884.

tion of the lateral columns means a division of fibres con-

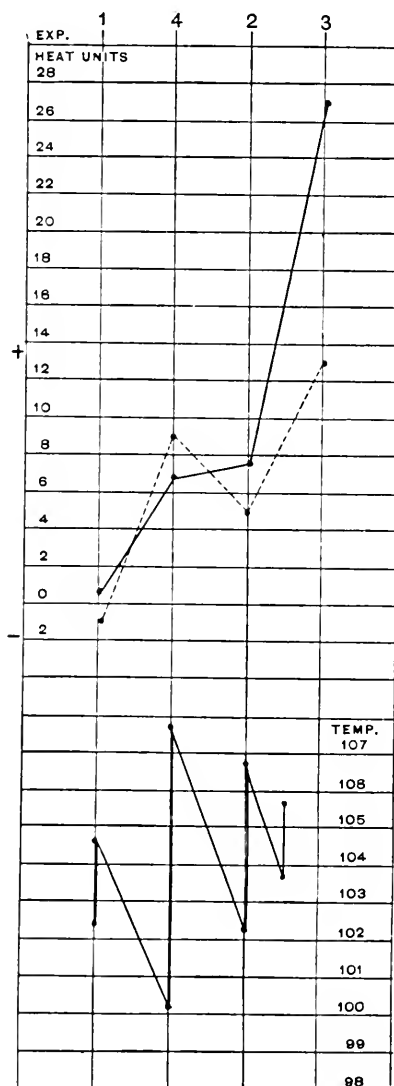


FIG. 1.

nected with centres in the brain which restrain the chemical changes in the protoplasm of the tissues.

*Decussation of the Thermo-inhibitory Fibres.*

It has already been noted by Schiff<sup>1</sup> after a hemisection of the oblong medulla at the level of the calamus scriptorius that a rise of temperature ensues in the neck, shoulders, seventh and last ribs, and the loins of the side opposite the section. On the side of section, there is a rise in the ears, lower eyelids, nose, and the anterior and posterior extremities. We have tried the experiment of injuring the heat centre on one side of the cerebrum, and noting the temperature on both sides. We found it, however, to be the same. We did note, however, when we divided the sphincter inhibitory fibres, thus causing a rhythm of the sphincters, that a rise of temperature always ensued. Now one of us, Dr. Ott, has shown that they run in the inner half of the middle third of the lateral columns, and they decussate. For that reason we believe that the thermo-inhibitory fibres also decussate, and it is possible that different inhibitory centres in the brain, either thermo- or sphincter-inhibitory, send impulses down the same fibres in the oblong medulla and spinal cord. For Dr. Ott has shown that the sphincter-inhibitory centres lie in the head of the crura cerebri and the parts of the optic thalamus adjacent to them, whilst the heat inhibiting centres lie considerably more anterior to these. We believe the thermo-inhibitory apparatus may be outlined as in Fig. 2. The fibres are connected with centre about the corpus striatum (1), also a point (2) between the corpus striatum and optic thalamus, which Schiff has pointed out as causing upon injury a peculiar cry, and the anterior end of the thalamus (3), whilst they decussate about the nib of the calamus in the oblong medulla (4), and pass down the lateral columns (5). The cortex of the cerebrum is (6).

We believe the body is maintained at a constant temperature by means of the sensory fibres, which call into activity the thermo-inhibitory centres which send influ-

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<sup>1</sup> "Untersuchungen zur Physiologie des Nervensystem," 1885.

ences down the lateral columns of the spinal cord, depressing the thermo-genetic centres, which are also in communication with the sensory system, but are so regulated that excessive heat, whilst it may act on the spinal heat generating centres, also acts with much greater force on the cerebral heat inhibitory centres, and thus checks heat production, whilst the absence of heat (cold) allows the sensory stimulation of the inhibitory centres to be less, and thus permits increased production. In other words, the action of the different temperatures on the sen-

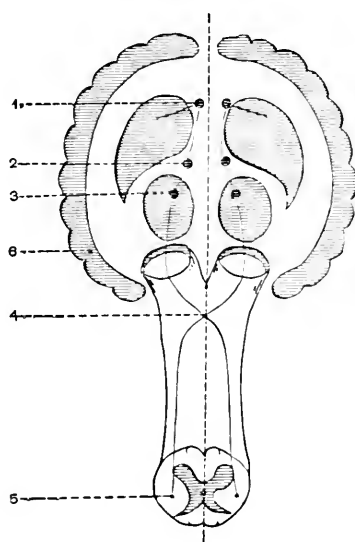


FIG. 2.

sory nerves, calling into more or less activity the thermo-inhibitory and spinal thermo-genetic centres, regulate the temperature of the body.

#### *Compression of Carotids.*

It is well known that when the carotids are compressed so as to interrupt the circulation in the brain, then the medullary vaso-motor centre is stimulated, causing a rise of arterial tension. We desired to see what effect it had on the thermo-inhibitory centres. It certainly did not call



them into activity, for there was a slight rise of temperature.

*Irritation of a Sensory Nerve.*

When a sensory nerve, say the central end of the sciatic, is irritated, the temperature rises temporarily and then falls. In an experiment made with the calorimeter, the sciatic during the second hour was irritated by means of shielded electrodes four times during the hour for a minute. The calorimeter is so arranged that electrodes can be inserted through the aspirating tubes, or even artificial respiration can be carried on in curarized animals without interfering with the accuracy of the experiment.

The results of our experiments were as follows ;

Heat dissipation during the first hour, 38.59.

Heat dissipation during the second hour, 29.20.

Heat production during the first hour, 37.69.

Heat production during the second hour, 28.37.

This experiment proves that heat production and heat dissipation fall as the temperature does from irritation of the central end of the sciatic.

Appended are some of the experiments. R. T. means rectal temperature ; A. T., air temperature ; and C. T., calorimeter temperature.

Exp. 1, Cat.

Time.	R. T.	A. T.
1.45 P.M.	101.6	104.5
1.55	One lateral column divided, part of the other lateral column and the posterior columns.	
2.08	101.5	100
2.13	101.8	Slight sphincter rhythm.
2.45	101.4	
3.35	99.3	98
4.20	100.6	100
4.45	101.6	101
5.10	102.1	101
5.35	102.3	99

Exp. 2.

Time.	R. T.	A. T.
3.10 P.M.	101.3	98
3.25	Gray matter and part of lateral column adjacent to it divided ; also posterior columns, sphincter rhythm marked.	
3.55	101.7	99
4.15	102.2	100

4.35	102.4	
5.05	103.1	101
5.30	103.0	100

## Exp. 3.

Time.	R. T.	A. T.
1.30 P.M.	102.6	102
1.50	Gray matter divided.	
2.02	102.2	102
2.35	101.8	101
3.00	101.6	101
3.30	101.4	99
3.55	102.	95
4.30	100.8	92

## Exp. 4.

Time.	R. T.	A. T.
2.00 P.M.	101.	102
2.08	Gray matter divided and part of right lateral column.	
2.15	101	101
2.40	101.6	101
3.00	101.7	101
3.50	102.4	96
4.35	102	92

## Exp. 5.

Time.	R. T.	A. T.
12.35 P.M.	99.9	101
One lateral column cut, the other partially; sphincter rhythm good.		
12.50	100.1	100
1.35	102	101
1.50	102.3	102
2.20	102.9	102
2.40	102.6	99
3.00	102.8	99

## Exp. 6.

Time.	R. T.	A. T.
1.00 P.M.	100.3	100
Gray matter divided; no rhythm.		
1.15	101.7	
1.40	102.1	102
2.10	102.3	101
2.30	101.5	99

## Exp. 7.

Time.	R. T.	A. T.
1.30 P.M.	103	98
1.50	Posterior columns and part of one lateral column.	
2.35	103.4	100
3.10	104.8	104
3.35	105.2	100
4.22	105.4	100

## Exp. 8.

Time.	R. T.	A. T.
2.00 P.M.	101.6	103
2.10	Whole cord divided, except a few fibres of lateral column.	

2.30	102	104
3.13	100.2	104
3.40	101.2	100
4.25	101.3	100
Exp. 9.		
Time.	R. T.	A. T.
2.00 P.M.	102	100
2.25 Lateral columns divided.		
2.45	103.2	95
Exp. 10.		
Time.	R. T.	A. T.
2.15 P.M.	100.2	100
Section of posterior columns, and injury of gray matter and a lateral column.		
2.55	101.5	100
3.25	101.9	100
3.55	102.4	99
Exp. 11, Cat.		
Time.	R. T.	A. T.
3.15 P.M.	102.3	98
Post columns divided.		
4.30	102.2	96
5.10	101.5	102
5.25	101.8	101
5.40	102	100
Exp. 12, Cat.		
Time.	R. T.	A. T.
2.35 P.M.	100.4	100
3.00 Anterior columns of spinal cord divided.		
3.20	100.8	100
3.50	101.2	99
4.45	101.3	97
Exp. 13, Rabbit.		
Time.		
1.46 P.M. Right side of body subcutaneously,	103	
Left side of body subcutaneously.	103 $\frac{2}{3}$	
2.22 Puncture into thalamus.		
3.14 Left side subcutaneously,	104 $\frac{1}{2}$	
3.15 Right side subcutaneously,	104 $\frac{1}{2}$	
4.10 Left,	104 $\frac{2}{3}$	
4.11 Right,	104 $\frac{2}{3}$	
Exp. 14, Rabbit.		
Time.	R. T.	
2.15 P. M.	102.5	
2.45 Both carotids exposed.		
3.25	100.8	
3.25	100.3	
3.30 Clip on both carotids.		
3.40	100.8	
3.55	101.2	
4.15	101.6	
4.35	101.2	
4.40 Both clips removed.		
5.10	102	
5.40	102.8	

## Exp. 15, Rabbit.

Time.		R. T.
2.15 P.M.		102.7
2.30	Both carotids exposed.	
2.45		100.8
3.10		101
3.40	Clips on both carotids.	101
3.55		101.3
4.05		101.3
4.20		101.2
4.35		101.3
4.50		101.2
5.10		101.3
5.15	Both clips removed.	
5.30		101.6
5.45		101.8
6.00		101.8

## Exp. 16, Cat, weight, 3.68 lbs.

Time.	C. T.	R. T.
2.45 P.M.	63.975	102.6
3.45	64.9	102.3

## Second hour.

Time.	C. T.	R. T.
4.00 P.M.	65.	102.9
5.00	65.7	102.3

Irritation of central end of sciatic by Du Bois coil.

## SOME NOTES UPON THE TREATMENT OF INSANITY.

By J. CHALMERS DA COSTA, M.D.,

LATE ACTING ASSISTANT PHYSICIAN IN PENNSYLVANIA HOSPITAL FOR THE INSANE, AND  
FORMERLY FIRST ASSISTANT PHYSICIAN TO THE INSANE DEPARTMENT  
OF THE PHILADELPHIA HOSPITAL.

A MAJORITY of the insane present evidences of physical ill-health, which condition has frequently preceded the attack of mental disease, and all who present them are materially benefitted by a course of medication aimed at this state. It seems highly probable, when we consider the many connections, marked sympathies, and far-reaching influences of the brain, that no person laboring under mental disease is in absolutely *good* bodily health, and though the impairment may be obscure, may be masked by the mental trouble, or not found through carelessness, it still exists. The physical ill-health may have caused the insanity, by inducing vaso-motor disturbances and qualitative or quantitative anæmia, and hence malnutrition of the convolutions. It may have been induced by the mental disease; as the brain, healthy or diseased, acts on every organ and tissue of the body, and when so induced reacts on the brain unfavorably. It may be an accidental complication. But in any case it *should* be sought for, and whenever found *must* be treated.

### CONSTIPATION.

This is a common prelude and a usual attendant of insanity. It is often due to a lowered tone of the nervous system, comprehending deficient secretion and impaired contractility of the muscular coat of the bowels. In subjects predisposed to insanity, obstinate constipation seems

to act as an exciting cause. Constipation produces gloom in a healthy man, and nearly all melancholiacs are less depressed and maniacs less excited after a free movement from the bowels.

When constipation exists, we begin treatment with the administration of an ounce of castor oil or grs. v. of calomel, followed by a saline, and after a free purge employ some medicine regularly, if required, to insure a daily movement of the bowels. The best remedy for this purpose is ext. cascara sagrad. fl. at night, in doses of ℥xl. to i. which will usually move gently in the morning. The elixir rhamni frang. (or buckthorn bark) is valuable in this way. If the patient will take pills, the following are admirable:

℞ Ext. belladon. ....	grs. ij.
Ext. physos. ....	grs. iij.
Ext. nucis vom. ....	grs. iij.
Oleoresini capsici. ....	grs. iij.
Aloin. ....	gr. i.
Ft. pil. no. xij. Sig. One or two at night.	

The belladonna and physostigma promote secretion and peristalsis; the nux vomica stimulates the muscular layer of the bowel and the central nervous system; the capsicum prevents griping (for this we may substitute ol. caju-puti), and the aloin acts upon the lower bowel.

Epsom salt, though valuable occasionally, should not be used continuously, as it promotes anæmia and hence weakens the individual.

Enemas are occasionally used, but not continually, as they do not sufficiently empty the upper bowel, do not act on secretion, and their administration is apt to create unpleasant delusions.

A most important means for the relief of habitual constipation is exercise in the open air, and another is diet. We should discontinue medicine for this purpose as soon as we find these means are sufficient. Out-door exercise promotes appetite and also peristalsis. As to diet, we should give fruit and green vegetables, regulate the amount of meat, give oatmeal, brown bread, and

prunes, and persuade the patient to *suck* an orange *before* breakfast. It will not do to cut down the amount of milk, and if it does constipate we will have to keep on using medicines. Kneading the abdomen will be found occasionally to antagonize the constipation from milk, without using medicines. We must not purge too freely or too often, as it weakens the patient very much, and this is especially true of salines. Clouston insists that a white tongue must not be considered an imperative indication to purge, as it may arise from want of food, depressed nervous energy, etc.

It is a good plan to construct a mixture having mild laxative properties, and containing some iron to combat anæmia, some nux vomica as a gastric and nervous tonic, and tonic doses of quinine. The following is useful:

R Magnesii sulph.....	℥ ss.
Ferri sulph.....	℥ ij.
Quin. sulph.....	℥ ij.
Strychnine.....	gr. ss.
Acid. sulph. dil. ....	f. ℥ i.
Aquæ .....	f. ℥ iv.
M. Sig. F. ℥ i. in water, t. in d.	

In this preparation the amount of saline is too small to prove injurious.

#### THE DIARRHŒA OF CONSTIPATION.

Many patients on admission to the hospital are found to be laboring under an attack of diarrhœa which astringents will not check. There will be a number of small watery, often nearly colorless, stools voided, the patient will be much prostrated, will complain of abdominal uneasiness or pain, headache, giddiness, dry tongue, and irritable stomach. This condition is due to a paretic condition of the bowel, causing the retention upon the sides of the tube of hardened fecal masses; the liquid portion passing through, and is most common in those cases which have been upon a course of opium. This condition is designated the diarrhœa of constipation, and is amended by several doses of castor oil and laudanum, Epsom salts

and sulphuric acid, or calomel and opium, producing several large stools, after which the diarrhœa will usually cease. In this complaint kneading of the abdomen does good, and stimulants are sometimes indicated.

#### INDIGESTION.

This induces gloom in a person mentally sound, and it never fails to aggravate the mental condition of a lunatic. Acid indigestion, due to fermentation, is quite common. An attack is relieved by an alkali after meals; but to cure, we use an acid before meals, and after meals one drop of carbolic acid, with grs. x. of subcarbonate of bismuth in aq. ment. pip., and mucil. of acacia (Bartholow). We must also exclude largely the starchy, saccharine, and fatty elements of the food. Tinct. nucis vom., gtts. x. before meals, t. in d., is very useful in preventing flatulence.

Atonic dyspepsia is much benefitted by f.  $\frac{3}{4}$  ss. of whiskey before meals, and acid hydrochlor. dil. and pepsin after meals. Irritable stomach, with nausea and vomiting, is cured by saline mineral waters, drop doses of Fowler's sol. before meals, or the above-mentioned mixture of bismuth, carbolic acid, and peppermint water.

#### THE USE OF STIMULANTS.

Whiskey and brandy are employed in conditions of exhaustion, and, as a rule, abandoned when this condition ceases. Most cases of melancholia are distinctly improved by stimulants in small quantities, being by their aid reacted from exhaustion, roused from lethargy and gloom, given sleep, better appetite, and improved digestion. When the excitement of mania is prolonged and exhaustion arises, stimulants are very valuable. Alcohol will occasionally prevent frenzy in melancholia, and oppose suicidal tendencies. It must be used cautiously in these states, we being careful not to over-stimulate. As a rule we employ f.  $\frac{3}{4}$  ij. or f.  $\frac{3}{4}$  iij. of whiskey or brandy daily, combined preferably with milk or milk and eggs.



If given to secure sleep, a hot punch is the most eligible preparation.

When the exhaustion from mania is profound, in acute melancholia, and in maniacal delirium, stimulants in large quantities are indicated, ammonia being valuable in the form of aromatic spirits. When convalescence is established, stimulants are stopped, except occasionally a little claret for dinner, or a moderate quantity of some malt liquor.

Malt is by many highly esteemed, and probably would more properly be considered under the head of foods. It is a food, a tonic, and a promoter of appetite and digestion, and is administered after meals in the form of extract. Personally I am not inclined to think that it produces results as pronounced as have been claimed for it.

#### DIET.

It is of the most vital importance that cases of insanity be well nourished, and good feeding often stands between them and hopeless dementia or the grave. In many cases of insanity, the appetite is absent, and in many more it is capricious, though in mania and general paralysis they often eat voraciously. The tissue waste occurring during active insanity is enormous, and even when the appetite is fair the individual usually loses flesh. When secondary dementia has occurred, they may become obese. In active insanity give large quantities of milk; it is the very best of foods for them.

In mania, milk, two or three quarts a day or even more, eggs, stimulants if there is exhaustion, *moderate* quantity of animal food, and plenty of out-door exercise to promote appetite, digestion, peristalsis, and sleep. In melancholia, four to six quarts of milk in the twenty-four hours, giving part of it during the night. Give also eggs, fat-forming foods, chicken, fish, etc. Out-door exercise is indicated unless the case is exhausted, a regular life and not too much meat. Milk is our best food in any case, and even if it constipates do not stop it, but give something to keep the bowels open. Whenever the patient is exhausted, is

losing flesh rapidly, or is sleepless, give food during the night.

#### FORCIBLE FEEDING

Is not harsh but humane, as it often saves life or prevents hopeless dementia. We must employ it not only when the patient absolutely refuses food, but when an insufficient quantity is being taken. By holding a glass of milk to the lips and urging the patient, we may persuade them to take it. A china vessel with a long spout may be used, the spout being placed between the lips and liquid food poured in the mouth. An ordinary china teapot will answer for this purpose. The food in this case consists of milk alone, or variously combined with eggs, brandy, sherry, cream, etc. If this fails, employ the nasal tube. This apparatus has a central syringe-bulb of rubber, and a tube at each end. One end is introduced in liquid food, and the bulb filled; the other tube is oiled and carried along the floor of the nostril into the pharynx, and the food is then *slowly* pumped in. We do it slowly to avoid vomiting, and do not introduce much over a pint at one time. If the patient is eating nothing else, give three times during the day and once at night a pint of milk, two eggs, a couple of ounces of cream, possibly some brandy or sherry, and any medicine we desire to employ. If the patient eats *something*, we may have to use this method only once a day.

If this apparatus is not at hand, a rubber catheter, with a funnel in one end, will answer, the food being slowly poured in. This method is practised while the patient is held slightly reclining in a chair. We may fail to introduce the nose tube, and in fact some patients get a trick of pushing it into the mouth. If we cannot pass it, hold the mouth open with a wedge, and pass a larger tube into the œsophagus and feed through that, but the nasal tube plan is to be preferred.

#### COD-LIVER OIL

Is a valuable restorative agent. Phthisis is common among the insane, and it is our duty to combat body-

wasting, which favors the development of this disease. For this purpose oleum morrhuæ is the best agent we possess. It is not only used in incipient phthisis, but is also valuable in cerebral softening, atheroma, etc. It is given in drachm doses, about two hours after meals, so that it will be subjected to duodenal digestion. Large doses are not well assimilated, and when we give them, oil can usually be found floating in the stools. If the oil is not well assimilated, give with each dose  $\mathfrak{m}\text{v}$ . of ether, which, by stimulating the flow of pancreatic juice, will favor its emulsion, saponification, and absorption. If sick stomach arises from the use of the oil, combine with it a small quantity of strychnine. An emulsion is a good form in which to give oil (Borell's emulsion).

#### PHOSPHORUS

Is employed occasionally in secondary dementia, in chronic mania, melancholia, and conditions in which atheroma exists. Bartholow considers its chief use is found in conditions of cerebral anæmia, local or general. Phosphorus alone is not often used; it is apt to disorder the stomach, and its prolonged use is attended with the danger of fatty degenerations. It is usually given in pill form, each pill containing  $\frac{1}{100}$  gr., and gr.  $\frac{1}{25}$  being given t. in d. A tincture is occasionally employed. Its best results are obtained in incipient insanity and in secondary dementias.

*The Com. Syr. of the Hypophosphites* has been highly lauded, but, though it does some good, it has been overpraised. It is of use in states of poor nutrition, and probably acts on the nervous system more by improving the general health than by specific selection.

The syrup calcii lactophos. also finds some admirers. With this I have had no experience.

*Acid Phosphate* is composed of the phosphates of lime, magnesia, potash, and iron. The iron is a valuable addition, and whereas I question the specific action of this preparation on nervous matter, it does benefit it some through improvement of the general system.

The *Pyrophosphate* of Iron is one of the best of the phosphate preparations when anæmia is an element in the case. Employ gr. iv. doses, t. in d., usually combined with a little quinine, in dil. acid phosphor. and aromatic mixture. I have found this preparation of considerable value in alcoholic insanity.

#### QUININE.

Clouston gives it the first position as a tonic in melancholia. In acute mania and chronic mania large doses are not employed unless there is some special indication. We give tonic doses (from grs. ij. to grs. vi. daily) as a rule, and it is of great use in exhausted and anæmic conditions. In puerperal mania it is used (in large doses), in secondary dementia, and during convalescence. It may with advantage be combined with strychnine and iron in many cases. Do not give too much quinine, as it may, like strychnine, convert a condition of depression into one of exaltation. The rule is *tonic* doses, except when we combat some special condition, as high temperature or chills.

#### ARSENIC

Is a very valuable restorative agent, as it promotes the nutrition of the brain, nervous system, and body at large. It promotes appetite, digestion, and intestinal movements. It forms a valuable addition to a tonic mixture, and is to be particularly esteemed in softening with atheroma, in incipient insanity, in dementia, and in convalescence. Vertigo is very common among the insane, and if due to stomach trouble, arsenic will be found curative. For this purpose, and for irritable stomach, Fowler's sol. is used. To the bromide mixtures used for epilepsy it is well to add a little arsenic; it tends to prevent bromism, and acts as a stomachic and general tonic. During a course of arsenic the patient should be carefully watched, and every few weeks we should intermit its use, and give a purgative and a diuretic. In incipient melancholia, Fowler's sol. combined with stimulant doses of opium is

often useful. Arsenic can be combined with advantage with iron, quinine, and strychnine, as in the following; A pill, to be taken t. in d., containing gr.  $\frac{1}{20}$  of arsenious acid, gr.  $\frac{1}{4}$  of ext. nucis vom., grs. ij. of quinine, and grs. ij. of ferri redact.

#### IRON,

Our sheet anchor of treatment in anæmia. In strumous conditions (which commonly exist in idiots and imbeciles) use it in the form of syr. ferri iodidi. Iron, quinine, and strychnine is a good combination, and in exhausted states, beef, wine, and iron is both beneficial and pleasant. Iron is used, for at least a time, in most cases of insanity, and is especially valuable in puerperal and lactational insanities, insanity with amenorrhœa, and with tendencies to phthisis. In giving iron, it is often necessary to also employ some preparation daily to obviate the constipation.

#### DIGITALIS

Is, of course, valuable in heart troubles among the insane, but its field of usefulness in the treatment of mental disease is small. In acute delirious mania, and in puerperal mania, large doses do good. It is of benefit in some general paralytics with weak hearts, and seems to have power in repressing the episodes of excitement in this disease. It has been held up as a valuable agent in stupor, but in this state I have seen no mental improvement which could justly be attributed to the drug.

#### NUX VOMICA AND STRYCHNINE,

Valuable as a nerve and stomachic tonic. May give alone or with iron and quinine. Strychnine is of the greatest value in melancholia and in secondary dementia, but must be used with care, as it may convert depression into excitement and produce circular insanity. Excitement is a contra-indication to the use of strychnine in any considerable dose. It is most useful in depressed and sluggish states of mind.

## NITRITE OF AMYL,

Valuable to prevent epileptic fits. Has been used in epileptic mania, sometimes with success, sometimes without. Spitzka praises it in stuporous melancholia, and in one case of this kind I saw it act wonderfully well. In stupor, about five drops are inhaled, t. in. d., watching its effects carefully, and stopping as soon as it causes very rapid action of the heart and marked flushing of the face. It is a very powerful remedy, must be used cautiously, and be only given after due consideration. It will often disappoint us.

## NITROGLYCERIN

Resembles amyl in action. Make a solution of one drop of nitroglycerin in 100 drops of alcohol. Start with a dose of  $\mathfrak{m}$  i. of this solution, and push up to  $\mathfrak{m}$  v. or more until we get the physiological action. It is used in epilepsy, epileptic mania, and stupor.

If we give either amyl nitrite or nitroglycerin, do not at the same time give either strychnine, digitalis, or ergot, as they are antagonistic. Be very careful in their use in stupor, as the heart in this state is usually weak, and they produce increased cardiac action, and a great fall in the blood pressure. I am inclined to think that we are not often justified in using them in insanity, except for the treatment of some bodily state, as angina pectoris or Bright's disease.

## BATHS.

Of course, employed at the dictate of cleanliness, but have other valuable uses.

The warm bath is very valuable in calming excitement in mania and melancholia. If there are indications of cerebral hyperæmia, conjoin with it cold applications to the head. The bath is used at bed-time, and should last from fifteen minutes to half an hour. The temperature should never be over 100° F., and it is better at 90° F. If it is very hot, or the patient is long retained in it, it may cause dangerous depression. These baths will

give sleep, promote secretions, and quiet excitement. Even when they fail to give sleep, they relax the system, and a small dose of some hypnotic will then often be efficient where large doses had previously failed. People with advanced heart trouble, or those profoundly exhausted, should not use them at all, or only for a short time. If much depression follows a bath, we must use stimulants. Cold to the head is often a valuable adjunct. We apply it by a rubber pipe, or by cloths kept wet with cold or even iced water.

In melancholia, short baths should be given daily for other purposes as well as securing sleep. The patient is rubbed down with coarse towels and alcohol, and the cutaneous circulation is thus stimulated.

The cold shower bath and cold bath must not be used, as they often hurry the patient into dementia. Turkish baths are highly praised by Clouston, and cold sponging, followed by friction, is often of use where there is sluggish capillary circulation.

Baths are only to be ordered by a physician, and should be supervised by him, as they have caused deaths.

#### CONIUM

Is not popular in this city. It has been much praised for its power in controlling muscular movements in acute mania. This it will do when given in considerable doses, by a paralyzing action upon motor nerves, but, though reducing motor excitement, it often increases cerebral. Just as in some epileptic insanities a violent muscular spasm serves as an outlet of nerve force which was before exploding on the psychic sphere, and stops the mania, so in acute mania the muscular movements are largely conservative, and to check them leads to increased mental trouble. Hence we should not stop them, unless temporarily, to effect a removal of the patient or in some other emergency, such as exhaustion arising from them, in which cases conium can be used.

Another bad result of the continued use of conium is that it destroys the appetite. In chronic mania it does no

good. I am satisfied that its administration should be the exception and not the rule, and that most cases of mania make an earlier and better recovery without it.

The combination of morphia and conium is, by some, held to be directly antagonistic to mania, but my experience with it is too limited to serve as a basis for correct judgment. In mania with chorea, conium finds warm advocates.

#### OPIUM AND MORPHIA.

In mania opium is not to be used, except as a very temporary expedient. If its use be continued in this state, it does harm, locks up the secretions, destroys appetite, hinders digestion, occasionally causes delirium, and helps to produce dementia. It usually takes a large dose in mania to produce sleep, and this may give rise to coma.

Opium is valuable in incipient melancholia in stimulant doses (℥ iv.–℥ v. of laudanum, t. in d.), and may substitute comfort for the mental depression. It is well to combine it with Fowler's sol., which prevents disorder of the stomach. At the same time feed well, take exercise, baths and friction, and secure sleep at night by chloral, bromide of sodium, or hot baths, and also use some laxative like cascara sagrada.

Opium or morphine is useful in melancholia to prevent frenzy, when we have a dilated pupil and soft pulse. They are best used in the day-time to secure quietness of mind, contracted pupils, etc.; sleep being secured at night by chloral, chloral and tinct. hyos., hot baths, etc. We must use a drug to prevent constipation, and as opium adds to the loss of appetite, we must insist on plenty of food, using forcible feeding, if required. We cut it down every few days as an experiment to see if frenzy returns. Stop the drug as soon as we can, without frenzy returning.

#### CHLORAL,

Our best hypnotic in many cases, and is useful in allaying excitement. It often procures from four to eight hours' sleep, and produces, as a rule, no unpleasant after-



feelings. It must be given carefully, as small doses may add to excitement, and large doses may cause dangerous depression of the heart. It should not be used continuously for any long period, as it weakens the brain and adds to dementia. It should not be used long in dementia, when the will is pronouncedly impaired, when there is weak heart, advanced phthisis, or when the patient is a drunkard. After using it for some time at night, the patient will occasionally be observed to be dazed, confused, and apathetic during the day, and when this is noted we must stop it. These symptoms are due to impaired brain strength, and if the drug is continued, pronounced and often incurable dementia will occur. Chloral should not be given continuously for long periods, and must not be given both day and night in the same case. To procure sleep, give grs. xx., and if need be, in one hour give grs. xx. more, but as a rule do not exceed this dose. We use it to produce sleep in any form of insanity, except to be careful to avoid much of it in dementia, or when the will is much weakened. It is used in epileptic mania, as an enema in status epilepticus, to quiet maniacs, and to allay the excitement of general paralytics. It occasionally disorders the stomach, but not nearly so surely as opium, and does not constipate.

A good combination, when there is much excitement particularly, is gr. xx. of chloral with f.  $\frac{3}{4}$  i. tinct. hyos., repeated if required. This often quiets and secures sleep when chloral alone fails.

Chloral is sometimes combined with bromide of sodium or potassium. This is dangerous to a weak heart. If we do use it, employ the sodium salt in preference to the potassium. A patient taking chloral must not be under the influence of strychnine, as these drugs are antagonists.

#### HYOSCYAMUS

Tends to allay excitement and produce sleep. Its A. P., hyoscyamine, is used hypodermically. It is used in mania with great motor excitement, and will give sleep and often

permanently stop the excitement. Much more valuable than conium. Must be used carefully, and not at all in those weak and exhausted. It is not suited for prolonged administration, as it gives rise to headache, dimness of vision, anorexia and may increase hallucinations. It sometimes fails utterly. It acts well in the excitement of paresis. It is combined with chloral to produce sleep and allay excitement in a great variety of cases.

#### URETHAN

Resembles chloral, but does not appear to depress the heart. It produces no stomach disorder. It is given in doses of grs. xx. to grs. xxx., and I have seen  $\bar{3}$  i. doses used in acute alcoholic insanity. It will often produce a quiet, refreshing sleep, but fails in power on repetition. There are no unpleasant after-effects. It is useful in mild melancholia, the premonitory wakefulness of insanity, and the excitement of idiocy, but fails in mania. Occasionally quiets in general paralysis.

#### PARALDEHYDE

In some cases acts wonderfully well, but in others appears almost inert. It is apt to seriously disorder the stomach, but may be given by the rectum with the yolk of an egg and mucil. acacia. By the mouth give  $\mathfrak{m}$  xxx. to  $\mathfrak{m}$  xl. in aquæ menth. pip., and by the rectum twice as much. It is suited to those cases which exhibit excitement with debility, hence to many cases in which chloral is not to be used. It is very valuable in some cases of acute delirious mania, and is best given by enema.

#### CANNABIS INDICA

Has a useful narcotic influence in many cases of excitement and depression. In small doses, in melancholia, it often rouses the patient from gloom and in large doses causes sleep. It is useful in chronic mania and may usually be combined with bromide with advantage in this state. The dose of the tinct. is from  $\mathfrak{m}$  x. to f.  $\bar{3}$  i., and we start with

the minimum dose and push up gradually. Hemp is well suited for prolonged administration, as it produces little or no disturbance of the stomach and other organs, neither does it predispose to dementia.

#### THE BROMIDES,

Given as pot. brom., sod. brom., ammon. brom., or a combination of them. Of high value to secure sleep in incipient insanity. Bromides are used in epilepsy and epileptic insanity for long periods. The trouble is that their prolonged use causes anæmia and gastric disorder, and predisposes to or develops dementia. Sod. brom. is the best salt and less likely to produce bromism. Do not use them continuously for long periods except in epileptic conditions. In acute mania the bromides alone will usually, and in chronic mania will often, fail to cause sleep. They are valuable often in epileptic mania, in simple delusional and hypochondriacal melancholia. After a hot bath has been given a case, a dose of bromide will often induce sleep.

A good combination to produce sleep is bromide of sodium or potassium, tinct. hyos. and ext. cannabis ind.

A combination with ergot is sometimes employed to combat epileptic mania and the maniacal outbreaks of paresis, and this combination is especially useful when great sexual excitement exists. Monobromate of camphor is valuable when this last-mentioned condition exists.

Blood-letting is not to be used for insanity. Antimonials are to be strictly avoided, and emetics are not employed.

#### HYOSCINE.

When I remember how much this drug has been praised, I am forced to consider it disappointing. The most extravagant claims have been made for it, but I am satisfied that, though of great value in some cases, its field of usefulness is limited. This statement is made with diffidence, as the weight of opinion is against it, but it is founded on

a number of observations, and at least in my hands the drug has not produced the magical results which some exhibit from minute doses. It is a most powerful agent, and has potent influences undoubtedly, but not always for good.

In acute mania it will induce sleep, but the sleep is not refreshing; in the morning the patient is exhausted, the pupils are dilated, the mouth is dry, there is muscular weakness, often tremor and inco-ordination, weak pulse, impaired or destroyed appetite, and often an increase of hallucinations

In chronic mania, *small* doses will for a short time cause sleep, but very soon larger doses are required, and then we get the results noted above. Again, small doses not infrequently add to excitement.

I have seen cases of different forms of insanity, who had been taking for some time hyoscine by the mouth, the doses varying from gr.  $\frac{1}{40}$  to gr.  $\frac{1}{10}$  at night, who in the day-time exhibited some or all the above-noted unpleasant symptoms, have the drug entirely withdrawn, sleep as well or better, improve in appetite, and get rid of the other disagreeable features of its action.

In melancholia, I believe, its continuous use almost always does harm by weakening the will, impairing the appetite, increasing exhaustion, stimulating the production of hallucinations, and adding to gloom. It is useful for a time in violent mania in apparently robust people, in whom there is strong pulse and contracted pupils. It may produce sleep in this condition when other means fail, but even here its employment must not be continuous for any great length of time.

It is very efficient in any form of insanity, particularly if given hypodermically, in producing quiet, and hence the temptation to use it is strong on the ground of personal convenience, but do not use it so. Only employ it when there is some special indication.

In attacks of great violence, when we use only one or two doses, it is of the greatest value. It is so used with great advantage in epileptic mania, transitory mania,

maniacal furor, and the excitement of paresis. It is useful to give an occasional dose in ordinary mania to quiet destructive tendencies, but its use must not be prolonged. It may be used for a short time in melancholic frenzy, and when there are uncontrollable impulses towards suicide, to gain time for better measures. It is also useful when we wish to move a case.

The dose varies, particularly because of the fact that there are impure imitations of Merck's preparation upon the market. We should be sure it is Merck's. Start with a dose of gr.  $\frac{1}{200}$  by the mouth to test the tolerance, and can rapidly push up to gr.  $\frac{1}{80}$ . Hypodermically we do not, as a rule, use over gr.  $\frac{1}{120}$  of the pure article. I saw gr.  $\frac{1}{80}$  hypodermically produce unpleasant symptoms in a case of agitated melancholia, there being marked prostration following its use.

Hyoscine in full dose hypodermically produces sleep in from eight minutes to fifteen minutes, which sleep is of uncertain duration. In violent mania it is usually of only an hour or two duration, but in dementis it will often continue for many hours. I repeat that it is of great value for occasional use, but must not be given continuously for a long time. If it is long given it hastens dementia.

It is best not to regularly employ these narcotics, sedatives, and hypnotics; we must be driven to their use, and remember that the sleep they produce is not worth one-quarter of the same length of natural sleep. The best sleep comes from air, food, and exercise. Often we must use these remedies, as the patient would die without them, but we stop them as soon as we can do so with safety.

I do not pretend in this article to exhibit all of the resources of treatment, but have merely considered some of the most common. I do not wish to appear dogmatic, nor to seem oracular, and I contribute this paper with diffidence, knowing I may expect contradiction, and merely reserving to myself the right to be adjudged as honest in my convictions.

## Clinical Cases.

### REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILA DELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF CHARLES K. MILLS, M.D., VISITING  
PHYSICIAN.

#### CASE XV.—*Katatonía.*

The history of the following patient is imperfect, some of the records of the case having been lost at the time of the fire at the hospital, and it being difficult to obtain from the patient himself a clear and connected story.

The patient, a young man twenty-three years of age, was admitted to the nervous wards of the Philadelphia Hospital in 1883. He had been somewhat depressed mentally, although a clear history of melancholia could not be obtained. He was at this time distinctly cataleptic, and exhibited some of the phenomena of automatism at command. On one occasion, Dr. Mills gave to a ward class a demonstration of his cataleptic condition. Later he became excitable and somewhat violent, and was transferred to the insane department of the hospital, where he has since remained. At one time, he had hallucinations of hearing and hystero-epileptic and cataleptic seizures. He was and is addicted to the use of morphia. The records of his condition at the time he suffered from mania have been lost.

The following are the statements of his physical condition at the time of admission to the insane department:

Height, 5 feet  $7\frac{1}{2}$  inches; weight, 135 pounds; dark complexion; slender but well built and well nourished; head and face well shaped; no abnormality of organs of special sense; abdomen distended and prominent; prepuce long; patient active and no special disease of any organ. He was fairly educated and had studied and practised pharmacy. He stated that he had to discontinue his business because he heard strange noises and sometimes had spells.

A few records entered in December, 1883, have been preserved. The following notes were made December 28th, 1883: "The

patient is quite communicative, he shows a tendency to use grandiose and high-sounding words and phrases, without a strict adherence to their proper meaning. His expressions are also somewhat involved and halting or hesitating. The following is quoted as having been used by him at this time: "I was emphatically disposed to be absorbed in the occupation of attempting what I had purposed, etc."

Since 1883 the patient has remained in the hospital, making himself useful in various ways, as assistant in the ward, runner, carrier of the mails, etc. He has shown himself trustworthy, taking an unusual interest in the work assigned to him. His manner and conversation, however, are at all times peculiar and very difficult to describe. His voice is usually effeminate or slightly piping in quality, with a tendency to rising inflections. His expression is smiling or complacent, and his movements might be described as mincing or effeminate. If started upon a subject of interest to him, he will talk on and on, like one wound up for an indefinite period, with stilted, involved expressions, sometimes hesitating, sometimes repeating, using long, sonorous words or phrases, or curious periphrases, sometimes showing slight verbal incoherence.

The following are a few illustrations of his method of talking, taken by a stenographer during this month (June, 1887).

Speaking of the morning papers he said:

"It is one of the ways in which they so much represent the general matters in the item and circumstances—I believe concerning the worldly matters or in matters that would be important to us to receive—I presume everything in general that would come to a paper in the general sense of the term, that is the report of persons and misdoings and so on."

When asked his opinion of one of the patients he replied:

"He seems to be more or less afflicted, combined with slight delirium. He is noisy and troublesome in every way, but I believe he is rather harmless. He does not seem to inflict any one in any way. He seems to be very gentle. He does not offer or show any intention to hit any one, but rather he seems to be rather delicate in his way, but noisy, very noisy, and troublesome, too, sometimes."

When asked whether he could remember the details of his own case he answered:

"No, sir, I don't remember—I scarcely sometimes, but most of the time. This being around about, being in the wards, I believe I know in a general way what I have been engaged at—but no, sir—I scarcely remember the things. Everything seems to be more or less combined and so indistinct that I am not able to realize. In fact, I have more or less a leaning to weakness in some way—rather I do not seem to have strength of mind or greatness—that it seems more or less delicate and weak—slightly so from the effects of the strong minds."

## Society Proceedings.

### NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, March 1st, 1887.*<sup>1</sup>

*The President, C. L. DANA, M.D., in the Chair.*

#### PRESENTATION OF THE CORD AND NERVES IN A CASE OF ALCOHOL PARALYSIS—MULTIPLE NEURITIS.

DR. H. M. BRIGGS presented the case.

*Autopsy.*—Patient greatly emaciated. Legs and thighs markedly flexed. Muscles of the legs of a yellow color, and apparently converted almost entirely into fat. Muscles of thigh much less affected. Spinal cord, nerve roots, and trunks normal in appearance.

*Microscopical Appearances.*—Spinal cord apparently normal, with the exception of slight sclerosis in the columns of Goll in cervical region. Nerve roots normal. In one of sacral nerves before its exit from spinal canal was found a marked increase in the endoneurium with diminution in the number of the nerve fibres, and an irregularity and indistinctness in these appearances. The right sciatic nerve showed the same changes more marked. In the posterior tibial the process was even more advanced, and in this only an occasional nerve fibre could be detected. Microscopically, the gastrocnemius was composed almost entirely of adipose tissue; only here and there atrophied muscle fibres were found. The small nerve trunks in the muscle showed advanced degenerative neuritis, with comparatively little new growth of connective tissue in the nerves.

THE PRESIDENT thought that in this case it had been fully demonstrated that the alcohol paralysis was due to a neuritis and not to a myelitis.

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<sup>1</sup> By an unfortunate error, the publication of these proceedings has been unduly delayed.—ED.



DR. M. A. STARR had seen the specimens, and said there was no question with regard to the existence of neuritis in this case, and the normal condition of the anterior cells of the spinal cord. There was slight sclerosis in the columns of Goll which he was unable to explain. The same condition has been observed in a case of Hamilton, recorded by Granger Stewart. He referred to a well-prepared specimen by Dr. Van Gieson, in a case of Dr. Ball's, not yet published; also to the manner of preparing specimens.

DR. BIGGS said that, contrary to the ordinary condition found, the process seemed to be more a degeneration of nerve fibre than an interstitial neuritis, especially in the smaller nerves.

DR. NOYES spoke of the frequent occurrence of amblyopia with alcoholism, and said it was due to a partial neuritis of the optic nerve, referred as had been shown to the centre field, and not to the field at large. He suggested that in cases like the one reported by Dr. Biggs, the neurologists should make careful examination of the optic nerves. In reply to Dr. Starr, whether scotoma was due as frequently to tobacco as to alcohol, he said it might be due to either; but the patients frequently combined the two habits.

THE PRESIDENT said the name alcohol paralysis was rather begging the question; this patient, it seemed, had been only a moderate drinker. The same fact had been noticed in other cases.

Abstract of DR. STEVENS' paper on

IRRITATION ARISING FROM THE VISUAL APPARATUS CONSIDERED AS ELEMENTS IN THE GENESIS OF NEUROSES.

Two classes of influences were recognized as causes of functional nervous disorders—the remote and the immediate. The remote causes may be sufficient to perpetuate a neurosis when once a nervous irritation has been instituted. While immediate causes rarely induce long-continued disorders, a pre-existing influence may serve to continue it indefinitely. It is of little practical importance that some exciting circumstance has given rise to a nervous trouble. The event has passed and cannot be recalled. If there is an underlying cause it is of much more importance.

Persons in whom underlying causes of neurosis exist are said to possess a neuropathic predisposition, and those subject to it are liable, from trifling immediate causes, to suffer from various neuroses. In a considerable proportion of cases, the neuropathic tendency is hereditary, but the result is not always manifested in the same form in different generations.

A third class of cases which should be recognized may be designated as *modifying tendencies*, among which may be mentioned vitiated atmosphere, the period of life, and the performance of certain physiological functions.

Often as a result of the predisposing influence, when one form of complaint is supposed to be cured, the subject of it is simply suffering from some other form.

Must the predisposing cause of neurosis be general, pervading the whole organism, or must it of necessity be located in the great nerve centres, or may it be entirely local and outside those great centres? Undoubtedly it may be local, and confined to any portion of the nervous system.

Inasmuch as the tendency is often hereditary, may not the evil consist of some peculiarity of anatomical structure or of physiological adaptations which are inconsistent with the most regular and easy performance of the function of a part or parts; and may not certain classes of mechanical peculiarities be unusually liable to become factors of physiological disturbance?

If we answer in the affirmative, we assume a hypothesis which must be maintained by long-continued observations, conducted in a spirit of judicial independence, and free from the bias which might result from occasional and exceptional experiences. The conclusions announced this evening are based upon observations in more than five thousand cases in private practice, and of a considerable number in public institutions; all of which have been made with as much precision as the exacting demands of an active professional life would permit. The central truth, as arrived at by these observations, may be stated, as it has already been done in a memoir to the Royal Academy of Medicine of Belgium, in 1883, as follows:

Difficulties attending the functions of accommodating and of adjusting the eyes in the act of vision, or irritations arising from the nerves involved in these processes, are among the most prolific sources of nervous disturbances, and more frequently than other conditions constitute a neuropathic tendency.

In the proposition, all causes of nervous irritation are recognized. It is held that the influences indicated are pre-eminent, but not exclusive permanent causes. Let it be remembered that it has been universally conceded that the nature of the neuropathic tendency is unknown. If one pre-eminently important

element is demonstrated it is not to be rejected because it may not include the whole.

The speaker proposed only to illustrate the result of his experience by exhibiting some photographs of cases of notable neuroses, which showed very remarkable changes of physiognomy, such as habitually occurred when certain hurtful tensions of the ocular muscles were relieved. If he had designed to present only the most remarkable cases of the class to which these belonged, he would have chosen only a few of these. The design was, however, only to show by these contrasting photographs the very notable improvement which in obstinate, and even by ordinary means, hopeless cases of the most important neuroses might be expected from relief of certain hurtful tensions of the eye muscles.

The portraits were in pairs, the first having been taken at the commencement of treatment; the second at a later period, the intervals being on an average about one month. The first series represented cases in his private practice; the second series cases which were under his care for a short time at the Willard Asylum for the Insane last summer. The first series had been made by various photographers, the second by Dr. P. M. Wise, superintendent of the Willard Asylum. Thirteen pairs of photographs were exhibited, nine of the first and four of the second series. In all these very striking contrasts existed between the first and second portraits.

In No. 1, a weary and listless young girl, a sufferer from headache, and who had never been able to attend school, is seen to be transformed in twelve days into a vivacious and thoroughly awake child, following relaxation of each of the inner eye muscles. The change in health was marvellous. In No. 2, an epileptic girl, whose vacant gaze and half-open mouth indicated a profound degree of dementia, within a single month put on an appearance of robust health and of lively intelligence. In another case a boy, choreic from infancy and imbecile, whose constant movements were too rapid even for the modern photographer, showed in the second photograph, from which the distortions of the face and wrinkles in the skin had disappeared, a clever mental state.

The speaker related in brief some of the results of a short season among the most hopeless cases of the Willard Asylum for the Insane. Two of these hopeless cases, who had, during the month preceding, treatment directed to the eye-muscles, been

subjected to about one hundred and seventy convulsions, suffered only about forty convulsions in the month succeeding that treatment, notwithstanding the withdrawal of all bromides.

Fifty per cent of epileptics so treated had remained well for a length of time varying from many years to only a single year, but sufficiently long to indicate that a great change had been wrought. Thirty-two per cent had received very marked relief, but short of absolute cessation of the complaint. They were all better without drugs than they had formerly been with. In seventeen per cent no good results had been obtained.

The speaker thought that, with a better understanding on his own part of the extremely complicated conditions of the ocular muscles often found in epilepsy, this record could be improved.

The method of procedure in examining for muscular defects was given. It differed radically from that proposed by Graefe and generally adopted, and from other methods which had been suggested. His method of performing tenotomy was also described.

In conclusion, Dr. Stevens said he thought it was not unreasonable to look for the future advance in medical practice along two great lines; the one related to micro-organisms, the other to irregular phenomena resulting from well-defined causes of irritation, which causes must be sought for principally in the direction of difficulties in the performance of necessary functions. With the removal of such difficulties, we might look with confident expectation to the cessation of the peculiar irregularity which constituted the special form of nervous disease.

DR. E. C. SEGUIN said, with regard to the ætiology of neuroses and serious mental disorders, that he thought we ought to look a great deal deeper than the exciting and superficial causes which occurred in many cases of that kind. In epilepsy and chorea, for instance, he thought we had to look for the efficient cause, not in disturbed external apparatus, but to hereditary predispositions and faulty tendencies. That faulty external apparatus would cause more attacks, or possibly aggravate the mental disorder, he thought no one would deny, consequently the optic apparatus, the genital apparatus, etc., should be put in perfect order. As to the great improvement after tenotomy in epilepsy, the records of surgery and medicine were filled with cases in which trauma of various kinds had interfered with epileptic manifestations for months or even years. It seemed to him the report of a case

within six months after tenotomy was rather premature. He referred to one of his cases of epilepsy recently submitted to division of the ocular muscles, the bromides at the same time being withdrawn, and three days later she commenced to have from six to twelve convulsions in the twenty-four hours—more than she had ever had before the operation. He had had patients go three years without an epileptic attack, and then have a relapse.

DR. H. D. NOYES thought the precise ocular conditions in the cases reported should have been recorded; perhaps they were in that part of the paper not read. He had with him exact records of a number of cases of ocular trouble with the result of treatment. It had not fallen within his experience to meet with the class of cases referred to by Dr. Stevens. He dwelt upon the importance of making a thorough ocular examination, including that of the muscles of the eye in every case. He had come to realize more and more the importance of insufficiency of the external recti. He had obtained benefit in many cases from prisms. He spoke of the method of examination and of performing tenotomy. The paper deserved the most careful consideration.

DR. D. B. ST. JOHN ROOSA said that that part of the paper which especially concerned the ophthalmologist was as old as ophthalmology itself, and it did not call for discussion to-night. The real point in the paper was, he thought, that the correction of errors of refraction, improper relation between the ciliary and internal recti muscles, and other deviations of the ocular muscles, was capable of curing constitutional disease. He took it that epilepsy was a constitutional disease and not merely a functional disturbance. The same was true of chorea. The question was, did these operations cure epilepsy and chorea? But it had been shown that people with chorea got well without ever having error of refraction corrected. It had also been shown that the vast majority of people who were not myopes were hypermetropics, yet suffered no inconvenience from it. In this, the author's second paper, another step had been taken, namely, that these constitutional diseases, epilepsy and chorea, were due, not solely to errors of refraction, but to want of co-ordination between the recti and ciliary muscles. Then the prism test came upon the field, and we had to exercise the ocular muscles by prisms. Then in the order of advance came the doctrines taught in the paper of to-night. Granting the claims of the paper, that the patients had for a time after correction of an ocular difficulty been greatly re-

lieved, possibly cured, yet that was a long way from assuming that the ocular disturbance, whatever it was, was the cause of the epilepsy. Many great men having strabismus had not become choreic, epileptic, nor insane.

DR. A. L. RANNEY thought that following the exhibition of the photographs little need be said in confirmation of the views advanced by Dr. Stevens. The photographs were so startling that they would be accepted in any court of justice by an unprejudiced jury as proof that unmistakable benefits had been derived from the treatment. He had personally seen and examined several of the cases, and he considered the published histories as decidedly underestimated. Dr. Ranney had performed the operation for the relief of ocular insufficiency nearly two hundred times, and had carefully examined the condition of refraction and accommodation as well as that of the ocular muscles in several hundred subjects afflicted with various forms of nervous disease. He did not pretend to pose as an oculist, but as a neurologist. Originally he was a skeptic, but his skepticism became no longer tenable when he saw a choreic and epileptic imbecile in Dr. Stevens' office who was perfectly restored in a short time to health and mental sanity by the method he had described. He thought the paper would tend to establish a new era in neurology. Regarding the operation, in no case had he had bad effects from it, but the treatment required careful regard to detail.

Respecting the view that the eye is an important factor in creating and prolonging the so-called "neuropathic predisposition," the following facts were pertinent: 1, No one has yet shown in what this predisposition lies; hence, if Dr. Stevens has shown that eye-defect is an important element in these conditions a great advance has been made. 2, There is no recognized pathology in functional nervous diseases. 3, Heredity is very common in these affections. 4, My records, in common with those of Dr. Stevens', go to show that eye-defect is found in a very large proportion of such subjects. 5, Many of the eye-defects found can be shown to be congenital, being inherited like feature. 6, The manifestations of the neuropathic predisposition vary with each case, and are called forth often by trivial circumstances which are too frequently regarded as of great clinical interest.

In the treatment of the severer forms of functional nervous disease, for example, in chronic epilepsy, one radical cure without the aid of drugs offsets a thousand failures as a proof of the scien-

tific value of a discovery. Dr. Stevens had seven cases free from epileptic seizures for more than five years after tenotomy of the eye-muscles, and without the aid of drugs. This could not be explained by chance. Then the records of the Willard Asylum were hard to contradict.

During the past year and a half he had seen sixteen epileptics in private practice; in only one was no defect in the eye-muscles found. He had an opportunity to operate on the eyes in eight of the cases; three of these were cured; two had no fits for over one year. In the five cases still under observation the attacks had been lessened in all, drugs having been withdrawn. One had been reported by Dr. Stevens. In headache and neuralgia, he had had some very remarkable results from tenotomy of the eye-muscles; also satisfactory results in hysteria and hystero-epilepsy.

DR. HERMAN KNAPP said his practice had not brought him much in contact with people who had neurotic conditions, and most of those whom he had seen had passed into other hands. He was very much surprised to learn that there was so high a percentage of ocular difficulties in the patients Dr. Stevens examined in the asylum. He thought nervous people generally showed not one complaint only. Many people, especially young ladies who suffer from headache, etc., cease to complain after correction of a deviation of the eye-muscles, etc. He had listened with the greatest attention to Dr. Stevens, and he felt quite sure that his work was not only legitimate, but that it was highly promising. He was only afraid we would not be spared disappointment in that line of treatment.

DR. GRUENING said his experience had been very much like that of Dr. Knapp. He always examined for muscular defect, and said that when one placed a prism before the eye it disturbed binocular vision. For the correction of this apparent muscular defect an operation was performed, but the muscle was sewed to its original place, or the lateral attachment was not divided, and this was only the simulation of an operation. He had benefitted many patients by cylinders.

DR. STEVENS, in closing the discussion, said there was no suggestion in the paper regarding cures. He did not believe in cures. Take away the cause of the trouble and they got well. If the patients could not be said to be cured, it was still a very fortunate thing that they had got rid of their chorea, epilepsy, etc.

PHILADELPHIA NEUROLOGICAL SOCIETY.  
PHILADELPHIA MEDICAL JURISPRUDENCE  
SOCIETY.

*Joint Meeting, January 24th, 1887, DR. S. WEIR MITCHELL in the Chair.*

DR. E. N. BRUSH read a paper on

THE DUTY OF THE STATE TO THE INSANE.

In caring for her insane wards the State should seek those means which shall best accomplish—

1. The protection of the insane from themselves and from the cruelty and neglect of others.
2. The protection of its sane citizens from the violence of the insane.
3. The restoration of the insane to a condition of sanity.

I have placed these objects in the order in which they are usually enumerated, and in which the law usually regards them. In the order of their importance they should be reversed, for the restoration of the insane to a condition of sanity implies the accomplishment in the course of its attempt of all the others.

It is, I think, a fact which cannot be controverted, that the insane are best cared for and their recovery most surely accomplished in organized hospitals devoted to their special treatment. This is certainly true of those of the indigent and pauper class, who cannot afford to make the arrangements which would otherwise be necessitated, and I feel confident that the hospitals of the future will afford to all classes such accommodations that private care will be as unnecessary as it is often unwise.

To insure the proper treatment of its insane wards, it becomes the duty of the State to make such provisions that all its insane can find ready and proper treatment in its hospitals.

In England and Wales the census of 1880 returns, on the 1st of January, 71,191 lunatics, idiots, and persons of unsound mind. Of these, there were in county and borough asylums, hospitals,



and district asylums, 44,035 paupers, 3,513 private patients, and 655 criminals; in licensed houses, 1,084 paupers, 304 private patients, and 61 criminals; in workhouses (corresponding to our almshouses), 11,991 paupers; residing with relatives and others, 5,980 paupers, and 468 private patients.

It will be seen that of the 63,090 pauper insane, 45,219 (or nearly seventy-two per cent) were in hospitals or licensed houses, and but 11,991 (or about seventeen per cent) in the insane wards of workhouses, and these are, as required by the commissions of lunacy, only of the most quiet and harmless kind. Of the private insane, 7,385 in all, all but 468 were in hospitals or licensed houses.

Let us compare this with the condition of the insane in the State of Pennsylvania, as shown by the United States census of the same year. Of the 8,304 insane in this State, as enumerated in the census of 1880, but thirty-six per cent were in organized hospitals, eighteen per cent in almshouses, and forty-six per cent at home. Massachusetts has sixty per cent in hospitals, nine per cent in almshouses, and thirty-one per cent at home; New York, fifty-seven per cent in hospitals, eleven per cent in almshouses, and thirty-two per cent at home. Of all the large States, Pennsylvania had the smallest proportion of her insane in hospitals, and the largest in almshouses and at home.

Of all the insane in the United States in 1880, nine per cent were resident in this State. Of all the insane in hospitals but seven per cent were in Pennsylvania, while sixteen per cent of all those in almshouses were in Pennsylvania.

Here is a condition of affairs that may well attract attention. I am happy to say that since 1880 a larger amount of insane have found accommodation in the hospitals and asylums of this State, but the number is still far below what it should be. According to the second annual report of the Committee on Lunacy of this State, its hospitals afforded accommodation for but 44.5 per cent of the insane. As the hospital accommodations have not been increased, the percentage at this time is much smaller, owing to the natural increase and accumulation of chronic cases.

Having made suitable provision for the insane, the question of the legal admission and detention of this class is of next importance.

The majority of the enactments in force in the United States and, indeed, in England to-day appear to have been framed under a most remarkable suspicion that the citizens of the State were in

constant menace of being spirited behind the bars of an asylum and forever disappearing from the view of mankind.

So strong appears to have been the conviction that medical men were ready to conspire to commit their fellow-citizens to asylums for the insane, and that the medical officers of asylums were waiting to aid and abet them, that the law of this State to-day directs that each examiner shall see the patient separately from the other, thus debarring each and the suspected lunatic from the benefits of conjoined examination and consultation.

In New York State, the year 1878 marks the commencement of a series of most malignant attacks upon the asylum system of the State, resulting in no less than five legislative inquiries, which have, I am happy to say, resulted in a vindication of the asylum officials and the lunacy laws of the State from the charge of improper commitment to asylum or unjust confinement or treatment therein.

The results, as concerns the unfortunate insane, were not so happy. For seven years (1872 to 1878, both inclusive), the average percentage of cases admitted to the asylum at Utica who were insane a year or more on reception, was thirty-three and one-third. In 1879 it rose to thirty-five and one-half, and in 1884 it reached fifty-one and six-tenths, the average for the second period of seven years (1879-1885) being forty-five per cent of practically chronic cases.

The second, third, and fourth reports of the Committee of Lunacy of this State will show that from forty-two per cent, or a little over two-fifths of the admissions, for the past three years to all the organized hospitals and asylums of the State, seventy-three and two-thirds, or nearly three-fourths, of all the recoveries were drawn.

These reports will also show that fifty-eight per cent of the cases admitted to the hospitals and asylums were chronic cases.

What is the remedy? Primarily, it seems to me, the creation of a public sentiment in favor of hospitals and asylums. The removal as far as possible of the feeling of distrust and suspicion which now surrounds the subject is necessary to induce the friends of the insane to take advantage of the facilities offered them for treatment.

It is, of course, implied that the State shall furnish sufficient accommodations for its insane before asking its citizens to take advantage of them.

One great obstacle which lies in the path of early asylum treat-

ment is the objection which physicians have against signing certificates of insanity.

The law places upon them the onus of saying not only that the person examined is insane, but that he is a proper case for confinement. They are not protected in any way against the annoyance of suit for damages brought by unrecovered lunatics before ignorant and prejudiced juries. Many physicians in general practice, moreover, feel themselves without sufficient training and experience in the diagnosis of insanity to be able or willing, in the very earliest stages of disease, to take the responsibility of diagnosis and certification. To obviate this, all medical schools should make instruction upon insanity a by no means unimportant part of their medical course, and attendance necessary to graduation. I speak with all due respect for the knowledge and diagnostic acumen of my medical brethren in general practice when I say that, of one single form of insanity, one that is singularly regular and characteristic in its manifestations, I have not known a single case out of over two hundred and fifty to be recognized before commitment. I refer to paresis. The mental disturbance was appreciated, as evinced by the certificate, but the peculiar form wholly unrecognized.

The fault was not with the physicians. They had never had occasion or opportunity to receive instruction in these cases.

Not by any means the least of the duties of the State is to provide, by Commissioners or otherwise, such a system of inspection and visitation of all places where the insane are confined that their friends shall be afforded a means of satisfying themselves of the proper conduct of such places.

Such a Commission is or should be an educator of the people—a bulwark on the one hand against unjust charges concerning asylum and hospital management, and on the other against the improper care and custody of the insane.

Imbued with a right spirit, they are strong factors in the establishment and maintenance of a proper system of hospital care, as witness the course of the English and Scotch Commissioners, who have been a real help to asylum physicians instead of captious critics, and who have by their course aided very materially in promoting the prompt and proper care of the insane. So strong has become the opinion of the English Commissioners in favor of early treatment that when it was recently proposed to throw, by an amendment of the law, increased difficulties in the way of early

commitments, the chairman of the Commission resigned rather than by retaining office seem to sanction such a change.

The Lunacy Committee of this State is, I believe, endeavoring to emulate the example of its English prototype. Under our more recent laws, which in some respects are not harmonious in all their relations, and with few American precedents to guide it, and an incomplete asylum system to work with, the Committee may not in all things accomplish at once what might be desired. Its hands should be sustained, however, and time and wider experience may be trusted to work much good.

#### DISCUSSION.

DR. S. WEIR MITCHELL.—With reference to the question of joint examinations, I think that the law does not forbid such examinations. It only makes necessary a separate examination by each physician. The joint examination, with all the good that arises from it, may follow. I favor separate examinations. Then the physician makes the whole examination for himself, while in the joint examination there is apt to be too much reliance put upon the opinion of the man older in the case.

DR. M. O'HARA.—It appears to me that the present certificate is a very poor one. It debars many from making the examination who should have a right to do so. Any one connected in the most remote degree with an institution cannot sign the certificate. Many physicians cannot be induced to go on a certificate. They are not willing to undertake the risks of subsequent prosecutions for false imprisonment of the patient. There follows, as a necessary consequence, that the practitioners who are probably most conversant with the case—for instance, family physicians—refuse, and thus the patient is handed over to strangers who are willing to take chances. Thus an early care of the insane tending to cure is prevented by the State not protecting medical men who are acting conscientiously in the line of their duty and with their highest judgment. The State should protect a physician, or otherwise give over the whole matter to a judge and jury.

DR. T. S. SCHULTZ.—I should like to emphasize the point in this admirable paper which refers to the present lack of accommodations for those who are not paupers and yet are not in circumstances to pay the rates charged by private hospitals. The law requires that the State hospitals receive public patients to the exclusion of those who can support themselves when there is not room for all applicants. There are many patients whose friends can pay the moderate sum which is charged by the State hospitals, but not any more. So long as these friends refuse to be pauperized by throwing their small means into the poor treasury, they cannot secure any accommodation. And this their self-respect, which is most commendable, prevents them from doing.

It is, to say the least, unfortunate that such persons, who are often curable, should be crowded out of State institutions, which

they assist to support by their taxes, by idiots, imbeciles, chronic and criminal insane, all beyond the reach of curative treatment.

DR. H. C. WOOD.—I feel to-night like saying a few words in regard to the protection of a class of the community in which I have a personal interest, and among which I long hope to dwell; that is, the protection of the sane. This subject has been forcibly brought to my attention during the past week. While at an insane asylum, visiting a patient, the conversation turned on our lunacy laws, and the gentleman in charge stated that not long ago he had a patient liberated by the Committee on Lunacy who went home, was at once adjudged insane, and incarcerated in an asylum in another State. I happened to go from there to the house of a patient in this city, and found the family considerably excited. They told me that a friend who had been in an insane asylum for numbaer of years, whose brother had killed a man and had been acquitted on the ground of insanity, had been liberated by the Committee on Lunacy. Shortly after this I met a physician in consultation, and he told me that not long ago he had sent a patient to the insane asylum, and that he had been liberated by the Committee on Lunacy. The patient then came to his office and told the doctor that he had no grievance against him for sending him to an asylum, but that he had a divine monitor in his breast which told him that the doctor was wrong in the judgment of his case. I do not mention these facts to find fault with the Committee on Lunacy. It is far from my desire to leave such an impression. These occurrences seem to me to indicate some vital defect in the law under which that Committee acts. As I understand it, there is no one on the Committee of Lunacy who would be recognized as an alienist. The law should require some one having experience in this disease to be on the Committee.

Again, I have over and over had my attention brought to cases of dangerous lunatics at large in the community, from whom it seemed impossible to have the community protected. A year and a half ago I was consulted in regard to a gentleman who had attempted first to seduce, and, this failing, to rape, his own daughter, and was only prevented by her outcries being heard outside of the house and bringing assistance. I said that the man was undoubtedly suffering with general paresis, and told the physician in charge that it was his duty to do something to protect the community. The family refused to have the man restrained. The attempt on the daughter was followed by an attempt on a stranger, which also fortunately failed. Yet the man was permitted to be at large for months after this occurrence.

I have been present at a number of trials in which the defence was insanity, and I have never yet seen a case in which the plea of insanity was fairly justified in which it was not equally plain that the man, before the commission of the offence, clearly portrayed that he was a dangerous lunatic. It seems to me that the

law should provide that some one having authority should take hold of these cases.

THOMAS W. BARLOW, ESQ.—I wish to make a few remarks with reference to the accountability of physicians who sign certificates of insanity. The passage of such a law as would lessen such accountability would, in my opinion, be repugnant to the progress and spirit of the age, the progressive and humane spirit which in this State pervades the laws thrown around those deprived of their reason. For many years laws have been enacted which more closely protect the liberty of the people. In New York State, wives have been permitted to testify where their husbands have been accused of crime. In this State, recently, persons accused of any grade of crime have been permitted to testify for themselves. This has been the spirit which has pervaded all laws passed within recent years. Now, in my opinion, sir, not only should the physicians be held accountable for what they do; not only should two physicians separately examine the patient who is supposed to be of unsound mind; but if it were possible to throw around such persons a greater protection, I should favor any such enactment.

In reference to the remarks of the gentleman who has just taken his seat, I would ask whether he himself knows or does not know if the woman who was recently released from an insane asylum, and whose brother was acquitted on the ground of insanity, is now a sane or an insane woman? This is the gist of the subject. He gives no instance of her present insanity; he has made no examination of her mental condition. He does not give us the weight of his authority, which is a most excellent one. It seems to me that, for his reasoning to have any weight, we should also have the weight of his opinion. In one instance in which the gentleman was an expert for the defence in the case of a man on trial for murder, the prisoner, four days prior to his execution, threw off the mask and admitted that he had been playing upon the credulity of his friends and physicians.

DR. H. C. WOOD.—I did not give the weight of my opinion in reference to the cases to which I have referred. They were simply things which had called my attention to this subject. I do say that, as far as I know, there is no way, according to the laws of Pennsylvania, to protect the community from a dangerous lunatic if his friends decline to interfere. I have seen numerous case, one of which I cited, in which criminal assaults were at all times imminent. In regard to the case to which the last speaker has referred, we have often discussed it. The evening is short and the papers are many, and I do not propose to be drawn into a discussion of it to-night. I followed that man nearly to the gallows, and saw no reason to modify my opinion. In the last days of his life he was in that mental condition in which, under skilful leading questioning, he could be induced to assent to almost any proposition that was made to him.

THE  
Journal  
OF  
Nervous and Mental Disease.

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Original Articles.

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TWO CASES OF HEMICHOREA ASSOCIATED  
WITH BRIGHT'S DISEASE.<sup>1</sup>

BY FRANCIS X. DERCUM, M.D.

INSTRUCTOR IN NERVOUS DISEASES, UNIVERSITY OF PENNSYLVANIA.  
NEUROLOGIST TO PHILADELPHIA HOSPITAL.

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THE following cases appear to be of unusual interest.  
They are briefly as follows:

CASE I.—F. B., a shoemaker aged fifty-eight, had for several years been under the care of the writer for chronic Bright's disease. At various times he suffered from headache and dyspnœa, and at others from marked mental depression. His urine had been examined many times and contained both albumin and casts. The former was always present, though the amount was never large. The casts were few in number, both hyaline and granular. They were only found after prolonged search. The quantity of urine did not seem to be affected.

Under treatment, the patient improved from time to time, being sometimes better and sometimes worse. For months together he would disappear from under observation, and at other periods the recurrence of his symptoms would again bring him under my care. However, from August to December of last year, his visits to my office

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<sup>1</sup> Read at thirteenth annual meeting of American Neurological Ass'n, July, 1887.

gradually increased in frequency, and on December 2d he presented himself with his dyspnœa much more marked and complaining of excessive headache. I ordered him home and intended visiting him on the following day. At midnight, however, I was hastily summoned by his wife, who told me that he had had what she termed a stroke. On arriving at the bedside, I found to my astonishment that his left arm and leg were being constantly thrown about by violent choreic movements, and that the left half of his face was being constantly distorted by grotesque grimaces. These movements were in no sense convulsive and were strictly limited to the left half of the body. The man was perfectly conscious, but he seemed dazed and stupid. A hasty examination showed that, while there was weakness of the affected side, there was no actual paralysis; and also that sensation was not materially impaired.

I was inclined to regard the case as possibly one of pre-hemiplegic chorea, but fortunately, as the sequel showed, decided not to use any very active measures. I merely prescribed a few large doses of bromide of sodium and concluded to wait. On calling the next morning I was surprised to find my patient down-stairs. He had, with some help, dressed himself and had insisted on going down. This feat he had accomplished with some difficulty; I now examined him with greater care. The choreic movements were less violent than during the night, though still excessive. They were still limited to the left side. The arm was the most affected, the face next, and the leg least of all. On comparing with the æsthesiometer the sensation of the affected side with that of the sound side, no difference was detected. The grip, however, was evidently weakened. The patient also complained of increased headache.

For two weeks following his condition remained about the same, though the hemichorea was on some days less marked than on others. Up to this time, digitalis and various supporting measures had been steadily maintained. In addition, bromide of sodium and chloral had been given



at night, as the choreic movements were at times so violent as to interfere with sleep.

On December 17th, I discovered that a large carbuncle was forming on the back of the neck. This new-comer rapidly increased in size and soon decided the fate of my patient. He grew weaker day by day. The chorea gradually became less and less marked, though it was never entirely absent. On January 2d he died.

On the day following I secured an autopsy, though this was limited to the brain. My notes are as follows: Calvarium somewhat increased in density. Amount of cerebro-spinal fluid estimated at five or six ounces. Dura thickened and adherent along the vertex. Sinuses and veins of pia much distended with blood. Meshes of the pia and arachnoid excessively œdematous, and exhibiting here and there milky opacities. Brain as a whole soft and also very œdematous throughout. Pia easily detached. Surface of cortex uniformly pale. Ventricles not perceptibly dilated. Walls of ventricles also excessively pale. Choroid plexuses somewhat cystic. Vessels of base somewhat thickened.

The substance of the brain was now exposed by horizontal section so as to reveal the centrum ovale, and the capsules and basal ganglia at various levels. Absolutely nothing beyond general œdema and pallor was noted.

Portions of the cortex were next removed for microscopical examination, as were also various blocks of tissue from the left hemisphere, especially the posterior third of the posterior segment of the internal capsule and contiguous portion of the optic thalamus.

As might have been predicted, this examination yielded negative results. Nothing abnormal was noted.

CASE II.—J. F., a carpenter, aged 60, stated that he had suffered more or less from vertigo for several years. In addition, he gave the following account of a rather remarkable experience: One day, two years ago, while walking upon the street, he suddenly lost power in his right leg, so much so, and so suddenly, in fact, that he fell

to the pavement and had to be assisted to a neighboring house. Strange to say, his paralysis lasted but a short time. It seemed to him but ten or fifteen minutes, though it may have been longer. At the end of this time he was able to use his leg sufficiently to walk home, though he did not regain complete power in it for several days. He asserts positively that he was perfectly conscious during this attack, that he did not fall by reason of vertigo, and that he was otherwise well. Further, he has had no recurrence of the attack.

Up to May of last year, he had, with the exception of occasional vertigo, enjoyed average health. At this time he was engaged in carpenter work upon a building, and upon one occasion while standing upon a scaffolding, he accidentally lost his footing and fell from the height of a few feet to the ground. He was somewhat jarred by the fall, but, with the exception of a slight bruise upon his right leg, he escaped without injury. Four or five days after the fall he noticed a slight twitching in the toes of the right foot. A few days later, this twitching had spread to the entire leg, and soon after the right arm became likewise affected. The trouble gradually increased until October last, when the jerking of the arm and leg became so violent as to interfere with sleep. He then presented himself at the University Hospital with the following symptoms. He had extensive and continuous choreic movements of the right arm and leg. The face was not affected, and inquiry disclosed that it had not at any time been involved. On testing the sensation with the æsthesiometer, no marked difference could be detected between the two sides. The grip of the right hand was slightly weakened.

His urine was examined and the examination frequently repeated at subsequent visits. It was pale in color, had a specific gravity varying from 1,010 to 1,015, and always contained a very large amount of albumin. It appeared, too, to be increased in quantity, as the man stated that he was obliged to rise two or three times every night to empty his bladder. Tube casts were found without very

much difficulty, though they were not numerous. There were small hyaline, large and small granular, and some fatty casts. The last were least frequent. There were also a few fat-drops and some compound granule cells.

The man was first placed upon remedies directed to the Bright's disease, but without any noticeable improvement in his condition. Finally he was placed, as a matter of experiment, upon very small doses of Fowler's solution, with a marked though temporary improvement. However, on account of the state of his kidneys, it was not deemed advisable to push the drug, and it was discontinued.

The condition of hemichorea was maintained without evident change until February of this year. The man at no time presented any symptoms of œdema of the face or limbs. The condition of the radial artery suggested sclerosis of the vessels. He at no time suffered from headache.

Up to February, the choreic movements had been strictly limited to the right side. Since that time, however, faint choreic movements of the left foot have been occasionally observed. The choreic movements of the right side are still violent, while in the left foot the movements are scarcely noticeable and are present only at times. The patient is still an occasional visitor at the hospital.

It may be of interest to append the report of an ophthalmoscopic examination made by Dr. S. D. Reiley. It is as follows: "The bearer has several granular spots in the macular region of the right eye, probably indicating incipient albuminuric retinitis. No other gross lesions."

How shall we explain these remarkable cases? Is there a causal relation between the Bright's disease and the hemichorea, or is the association of the two affections merely accidental? While I do not pretend to give a positive or final answer to this question, I maintain that the weight of evidence is in favor of the first hypothesis. In the first place, we have here no evidence of organic cerebral disease such as we are led to expect from the re-

searches of Dr. S. Weir Mitchell<sup>1</sup> and from the later work of Charcot<sup>2</sup> and Raymond.<sup>3</sup> There is in these cases no history of a previous hemiplegia, and secondly the autopsy of Case I. proved the entire absence of any focal lesion, while the most detailed and careful study of Case II. likewise negative any hypothesis of organic disease.

It may seem strange, indeed, that if the chorea be really related to the Bright's disease it should be so limited in its distribution. It may be difficult to understand why a general cause should give rise to such a localized set of nervous phenomena, and yet that disturbances of the nervous system strictly limited to one-half of the body do occur in Bright's disease is no longer a matter of doubt. In the *Revue de Médecine* of September, 1885, Raymond,<sup>4</sup> in a paper on certain paralytic accidents occurring amongst old people, and the probable relation of these accidents to uræmia, reports a series of remarkable cases, and among them five cases of sharply defined hemiplegia occurring in old men in which the autopsy revealed a general and marked œdema of the brain substance, *but not a trace of a focal lesion*. Raymond also demonstrated by actual experiment a one-sided action of the nervous system in uræmic poisoning. He ligated in an animal the hili of both kidneys after having previously removed the cervical ganglion of the sympathetic of one side. He did this in two instances, and in each case the uræmic convulsions which ensued were limited to one side of the body. Evidently in these experiments the power of the two halves of the nervous system to resist the action of the uræmic poison was made unequal by the mutilation of the sympathetic, and the result was a one-sided convulsion. This is, no

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<sup>1</sup> S. Weir Mitchell, *American Journal Med. Sci.*, October, 1874.

<sup>2</sup> Charcot, "Leçons sur les mal. du syst. nerv.," Tom. II.

<sup>3</sup> Raymond, "Étude anatom., physiolog. et clinique sur l'hémichorée, l'hémianæsthésie et les tremblements symptomatiques." Thèse pour le Doctorat en Médecine, 1876, Versailles.

<sup>4</sup> Raymond, "Sur la pathogenie de certains accidents paralytiques observés chez des vieillards. Leur rapports probables avec l'urémie." *Revue de Médecine*, Sept., 1885.

doubt, also the explanation of three cases<sup>1</sup> reported by Raymond in which there was a history of a former attack by hemiplegia with recovery, and in which years afterwards the occurrence of uræmia caused a reappearance of all the symptoms of the old hemiplegia without the production of a new focal lesion. The autopsies revealed cicatrices in one or the other hemisphere together with general œdema and nothing more.

Raymond does not stand alone in his observations. More recently Chantmesse and Tenneson<sup>2</sup> have reported four cases of uræmic hemiplegia and in which the autopsies revealed nothing but general cerebral œdema. They have also reported two cases of uræmic epilepsy in which the convulsions were strictly limited to one side. One of these cases was confirmed by autopsy, there being no focal lesion, merely œdema. The other case recovered from the convulsive attack, but afterwards suffered from variable and fugacious paresis of one or more limbs. This fugacious paresis is interesting in connection with the second case of hemichorea reported in this paper, in which there is a distinct history of a marked and yet transitory palsy of one leg.

It is important, also, as indicating that the one-sided action of the nervous system in uræmia is by no means limited to old persons, to note that three of the cases reported by Chantmesse and Tenneson were but thirty-five years old.

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<sup>1</sup> Cases V., X., and XI. Loc. cit.

<sup>2</sup> Chantmesse et Tenneson, "De l'hémiplégie et de l'épilepsie partielle urémiques," *Revue de Médecine*, Nov., 1885.

## HEMIPLEGIA IN CHILDHOOD.

By PHILIP COOMBS KNAPP, A.M., M.D. (HARVARD),

PHYSICIAN TO OUT-PATIENTS WITH DISEASES OF THE NERVOUS SYSTEM, BOSTON CITY HOSPITAL, AND PHYSICIAN TO THE DEPARTMENT FOR NERVOUS DISEASES, BOSTON DISPENSARY.

THE recent contributions of Strümpell (70, 71) on the subject of cerebral infantile paralysis have aroused a new interest in a symptom-complex which, although long recognized by foreign writers, has been much neglected by Americans and Englishmen. Although hemiplegia in childhood is often attended by certain peculiarities which entitle it to special consideration, none of the works on children's diseases, with the single exception of Money's (49) treatise which has just appeared, describes it or discusses the vexed question as to its pathology. Most of them speak of ordinary cerebral hemorrhage and embolism; but, with the exception of Money, none of them so much as mention the atrophy of the cortex so often met with, or give any full account of the special features of hemiplegia in childhood. This omission is the more singular, since many of the recent text-books on nervous diseases describe the affection with more or less fulness.

We owe to Cazauvieilh (14) the earliest description of that form of cerebral infantile paralysis which is now regarded as typical—the form due to atrophy of the convolutions—although he states that cases had been met with previously by his instructors in Paris. In 1853 Little (40), in England, in writing of “spastic rigidity of the limbs of the new-born” described cases of cerebral infantile paralysis, but he failed to distinguish these cases clearly from

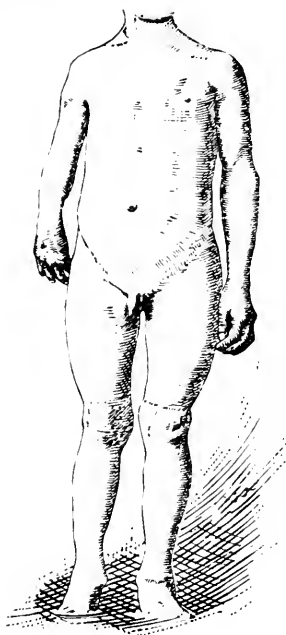
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<sup>1</sup> Candidate's paper read by title at the meeting of the Am. Neurol. Assoc., July, 1897.

cases of spastic paraplegia from spinal disease, or to recognize the pathological changes underlying the affection.

During the past year, I have had the good fortune to see no less than ten cases of paralysis due to cerebral disease in childhood. None of these cases have as yet come to autopsy, so that I can add nothing to the study of the morbid processes involved, but clinical study certainly gives some hints as to a possible pathology in the cases which I shall report.

OBSERVATION I.—*Convulsions in infancy. Right hemiplegia with contracture. Convulsions, now often limited to right arm. Arrest of growth on paralyzed side.*<sup>1</sup>



Frederick K., 11 years old, the second child of German parents. His mother had weak, faint spells before he was born, and is said to have uterine disease. With this exception the family history is good. One child died of heart disease, five others are living and healthy. There is no history of syphilis or hereditary nervous taint. He was born at term, the labor being twelve hours in length and rather difficult, but instruments were not used. The head presented. After birth no abnormality was noted except

<sup>1</sup> The illustrations are reproduced from photographs taken by Dr R. A. Kingman, of Boston.

blueness of the fingers. At the age of three months, he began to have convulsions, which gradually increased in frequency. When one year old, he began to get his teeth, dentition being difficult, and each tooth requiring to be lanced several times. The convulsions at this time grew worse, he had at times fifty or sixty a day, and sometimes ten or a dozen while his father was taking him to a doctor. Cutting the gums seemed to give relief. I could learn little as to the character of these convulsions except that they were general—he became rigid and all his limbs worked. Since dentition the convulsions have continued with more or less frequency. He has gone without any for two months at a time, and then he has had them quite frequently. March 26th, 1887, two days before he was brought to the hospital he had twelve, and the next day ten. The present convulsions begin with a feeling of something going down the right arm, which comes on long enough before the attack to enable him to go and lie down. In the severe attacks, everything looks dark to him; he can neither see nor speak; he cries out and works all over. Only in the severe attacks does he lose consciousness. Most of his attacks are confined to the right arm, and many of them to the right hand, and in these he is conscious and can speak. On May 23d, I saw him in a mild attack. There was no change of color in the face, and the right hand was the only part affected. He was perfectly conscious throughout and could answer questions. The pupils were not observed. The fingers of the right hand were strongly extended and abducted—a position which he could not put them into of his own will—and they twitched a little in adduction and flexion. The whole attack lasted forty or fifty seconds.

The boy could not walk until he was three years old, and then the parents noticed that the right side was much weaker than the left, a condition which probably had come on some time before. He now walks with a limp, but the arm remains partly paralyzed, and there is a decided arrest of growth on that side. He could not talk until he was three years old, and now his speech is rather indistinct. The trouble is purely one of co-ordination, for he never uses the wrong word or is at a loss for a word; he reads and writes as well as could be expected, and his speech is always intelligent. In spite of his hemiplegia and convulsions, his general health is very good; he never has the headache, eats and sleeps well, and has no trouble of any sort. His parents say that his teacher considers him a very good scholar, and they regard him as very bright, as bright if not brighter than their other children. Two years ago, he was sent to Dr. Baker's home at Baldwinsville for his convulsions, but he grew very homesick and stayed but six months. Since that time he has taken bromide—how much or how constantly I could not learn—without much benefit. Borax—fifteen grains three times a day—was given, and since then the convulsions have been fewer and less severe. An attempt was made to control the individual attack by a ligature at the wrist, but it had no effect.



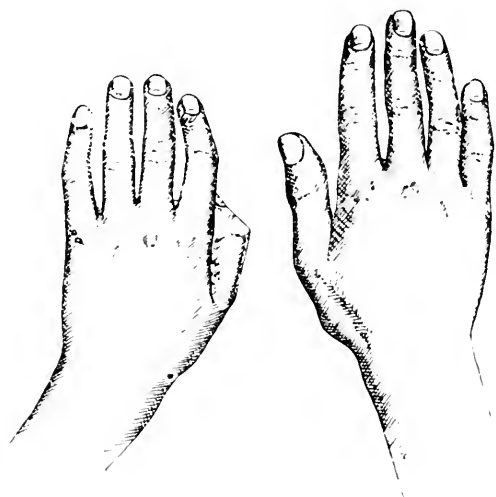
The boy is a little below the average height, but he is remarkably well developed, except on his paralyzed side, and well nourished. He appears very bright and quick-witted. The sense of smell is alike on the two sides. Vision is good; the eyes and pupils move naturally, and the field of vision and the fundus oculi are normal. The tongue is protruded straight. The right side of the face is smoother than the left, but he can draw his mouth better to the right; on whistling or laughing, the mouth is drawn a little more to the left than to the right. There is no difficulty in swallowing. The right side of the thorax is smaller than the left, but physical examination reveals nothing abnormal. The right arm is much atrophied, the hand is cold and cyanotic, and the arm is held against the chest, flexed at the elbow and wrist, strongly pronated, with the fingers flexed on the palm. The accompanying photograph, kindly taken for me by Dr. R. A. Kingman, of Boston, will show his ordinary attitude. Voluntary movements at the shoulder and elbow are possible, but he can neither supinate nor extend the wrist. The wrist can be flexed a little more, and there is a little movement of the fingers, chiefly in flexion. There is marked rigidity of the wrist on passive motion, and some of the elbow. The foot is in equino-varus, and the toes can be moved but little voluntarily; there is also marked resistance to passive motion at the ankle. The heel, however, reaches the floor on standing. There is no power of voluntary motion at the ankle, but motion at the hip and knee is fairly good. He walks fairly well, swinging his leg in the characteristic manner of a hemiplegic patient. The muscles of the arm reacted about alike on the two sides to a weak faradic current; except that on testing the extensors of the wrist and the supinators on the right the current had to be stronger, so that it was diffused, causing contraction of the antagonists, and the muscles tested did not respond as well. Sensation was everywhere normal. The epigastric, abdominal, and cremaster reflexes were normal; the plantar was normal on the left, but absent on the right. There was no triceps, radial, or ulnar reflex, or ankle clonus. The patellar reflex was exaggerated on the right, but very weak on the left, except by Jendrassik's method.

The following measurements were taken :

[illegible]

OBSERVATION II.—*Injury to head. Convulsions. Paralysis of left arm and face without marked contracture. Arrest of growth in arm.*

James E., 14, born in Italy. He was in good health until he was six years and a half old when he fell from a window, striking his head. He was unconscious for three hours, and then made a complete recovery. Three months later he had a convulsion. Since then he has had convulsions at various intervals, averaging about one a week. It was not easy to get a clear account of these attacks from either the boy or his family. He is said to cry out, bite his tongue, and lose consciousness, but he does not fall. I could not learn whether the attacks were preceded by an aura or not. In former attacks, the face was drawn to the left and the



left arm worked, but now neither the face nor the limbs are convulsed. Since having the convulsions, he has had loss of power in the left arm, which is now much smaller than the other. He has had no difficulty in walking, or paralysis of the leg, but lately he has complained that his legs ache, and that his left knee is sore. He has occasional frontal headache, but no vertigo. Lately he has had a little cough and palpitation and some pain in the chest. His appetite is good, but he has had a little diarrhoea for two or three months before coming to the hospital. He was given bromide of sodium, fifteen grains three times a day, which stopped his convulsions completely during the whole time that he was under observation—some five or six weeks.

The boy is well nourished and developed. The left arm is manifestly smaller than the right, and is cold and cyanotic. The eyes move naturally, the pupils respond to light, and the fundus

oculi is normal. He can perform all movements with his left hand except adduction of the little finger, but all the movements are feeble. The dynamometer showed a grasp of 46 with the right hand and 22 with the left. There is no contracture or rigidity of the arm, but the fingers are slightly flexed and are a little stiff and cannot be completely extended. The photograph, taken like all the rest by Dr. Kingman, shows this slight contracture, and the smaller size of the hand. The boy was directed to hold both hands up against a screen as high as he could, and, as will be seen, he could not reach as far with the left. He cannot draw his face to the left, either voluntarily or on laughing. No cicatrix or depression of the skull could be detected. There is no paralysis or noticeable weakness of the legs, which are of the same size. The fingers of the left hand are smaller and smoother. The muscles of the arms react alike to a weak faradic current. The triceps and radial reflexes are slightly more marked on the left. The patellar reflex is absent, except by Jendrassik's method, when it is very weak. Sensation is normal. Examination of the chest revealed nothing.

The following measurements were noted :

						RIGHT.	LEFT.
Circumference of arm	6 inches	above olecranon	.	.	.	7 $\frac{3}{4}$ in.	7 in.
"	"	3 "	"	"	"	7 $\frac{3}{4}$ "	7 $\frac{1}{4}$ "
"	"	5 "	"	"	styloid process of radius	7 $\frac{3}{4}$ "	6 $\frac{1}{2}$ "
"	"	1 "	"	"	"	5 $\frac{7}{8}$ "	5 $\frac{1}{8}$ "

OBSERVATION III.—*Right hemiplegia with contracture. Some mental impairment. Absence of convulsions. Marked arrest of growth on paralyzed side.*

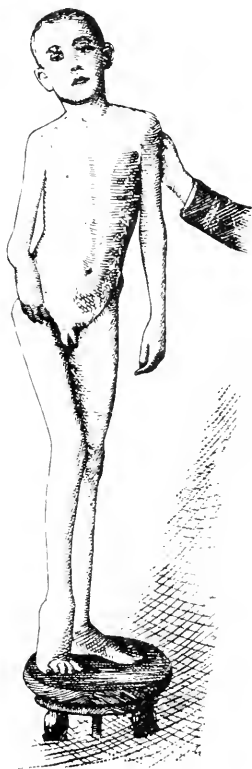
Wm. C., 11, of Irish parentage. He was born at term in one pain, the head presenting and the labor being natural and easy. When three weeks old he had scarlet fever mildly, and when five months old he had whooping cough. No history of any syphilitic or hereditary nervous taint could be obtained. He learned to walk at ten months, and after that walked and ran about like other children. About the same time he learned to talk. When a year and ten months old he was put to bed in his usual health, and nothing was heard from him during the night. The next morning it was found that he could not walk or stand alone, and later in the day his mouth was drawn to the left. Since that time the whole right side has been paralyzed, but there has never been any disturbance of speech. For three months he was in bed, but after that he got up and began to get about. At present he goes to school, and plays with the other boys, and in spite of his paralysis, can get over the ground pretty fast. Except for an attack of measles some years ago his health has been good, although he is not very vigorous. He began to go to school at the age of five, but thus far he has not distinguished himself. He is still in the primary school, and spent four years in one room. His teacher calls him a good boy but a poor scholar. He can read some and

write a little with his left hand. His father states that his memory is very poor. Until he was seven years of age his mother says that both sides of his body grew alike, but after that his right side did not grow as fast, so that it is now much smaller than the other. He has never had convulsions or *petit mal*, and is not subject to headache. He can talk perfectly well. There are no thoracic, abdominal, or urinary symptoms.

The boy is of the average size, rather poorly nourished, and presents a pronounced arrest of development on the right side, even the face seeming smaller on that side. He answers questions readily and comprehends what is said to him, but he is rather stupid. He is said to be able to read his school-books quite well, but on trying him with the printed diet list of the hospital he read the short words well enough, but he had much difficulty with the longer and more unusual words. The pupils and eyes moved naturally, the field of vision was good, and the fundus oculi was normal. The tongue was protruded straight. He did not wrinkle the right side of his forehead as much as he did the left in raising his eye-brows, but he closed his eyes naturally. He could draw his face to the right fairly well, but on laughing it was drawn to the left, and in repose the left naso-labial fold was more marked, and the left angle of the mouth was a little higher. This paralysis on mimetic but not on voluntary movement, seen here and in Obs. I., is of interest as an indication of the existence of separate channels for innervation. Rosenbach,<sup>1</sup> who has recently called attention to the subject, claims that the movements of expression are innervated through the optic thalami. The apparent paresis of the upper branch of the facial is remarkable in intracerebral disease. The accompanying photograph will show the atrophy and deformity. His right arm, which is much atrophied, is held to the side, flexed at the elbow and wrist, strongly pronated, and the fingers semi-flexed. The skin is cold and cyanotic. There is great resistance on attempts at passive motion, owing to the great muscular contracture; at the wrist this cannot be overcome by any safe expenditure of force. He can move the shoulder and elbow a little, but the only movement possible of the hand is further flexion of the fingers. The thigh is flexed somewhat on the pelvis and rotated inwards, and the leg is flexed on the thigh. The gastrocnemius is greatly contracted, drawing the heel from the ground, so that he walks on the ball and inner edge of his foot—the foot being greatly deformed, and in a position of marked equino-varus. This deformity cannot be overcome by any attempts at replacement. There is fairly good movement of the hip, some of the knee, but none of the foot or ankle. The pelvis is tilted to the left to counteract the deformity. There is a compensating lateral curvature of the spine which makes the thorax seem smaller on the par-

<sup>1</sup> P. Rosenbach, "Zur Lehre von der Innervation der Ausdrucksbewegungen," Neurol. Centralblatt, June 1st, 1886.

alyzed side, although it is not. He gets over the ground pretty fast, using the right leg as an occasional point of support, and hopping much of the way on the left foot. The muscles of the arm reacted alike on the two sides to a weak faradic current. Sensation was normal. Examination of the chest was negative. The abdominal and cremaster reflexes were normal. The plantar was normal on the left but absent on the right. The biceps, radial, and patellar reflexes were present, and were slightly more



marked on the right side. There was no wrist, patellar, or ankle clonus.

The following measurements will show the degree of wasting on the paralyzed side:

	RIGHT.	LEFT.
Semi-circumference of chest at nipples, . . . .	11½ in.	11½ in.
Circumference of arm 4 inches above olecranon, . . .	5½ "	6½ "
" " " 3 " " styloid process of ulna, 4½ "	4½ "	6¼ "
" " " 1 " " " " " 3½ "	3½ "	4½ "
" " thigh, 5 " " upper border of patella, 10¼ "	10¼ "	11¾ "
" " calf in largest part, . . . .	7¼ "	9½ "

	RIGHT.	LEFT.
Anterior superior spine of ileum to internal malleolus, . . .	23½ in.	29 in.
Length of ulna, . . .	6½ "	7½ "
Fingers of right hand and hand itself much smaller.		

OBSERVATION IV.—*Right hemiplegia of gradual onset. Temporary aphasia. Slight arrest of growth. No rigidity, contracture, or convulsions.*

Kate H., 12 years old, of Irish parentage. The family history shows no syphilitic or hereditary nervous taint. Eleven years ago she had diphtheria, three years ago whooping cough, and two years ago measles. Except for these illnesses she has been well until a year ago. At that time, on her return from school, where her work had not been especially hard, she had a "reeling in her head," and fell over on the floor. She was picked up and probably had some difficulty in walking immediately after. There was no loss of consciousness, but her right side gradually became paralyzed, and her face was drawn to the left. For six weeks she was confined to her bed, then she gradually got able to move about and to use her limbs, and since then she has steadily but slowly improved. She never had convulsions, but during her confinement to the bed she complained greatly of headache and heat in the head, and ice was applied to the head by her physician's orders. Six months ago she had some twitching of the right hand, which has disappeared. For three months she could not talk so as to be understood, but now she talks without any noticeable defect. During the first of her illness she was rather slow and dull in her mental processes, but now her mental condition is natural. At present she has some headache, chiefly frontal or in the left temporal region, and a little vertigo. There never have been any thoracic symptoms, and before this attack she could run and play like other children. The digestive functions are performed normally. She sleeps well, and there has been no change of disposition.

The pupils and eyes move naturally; the field of vision and the fundus oculi are normal. She cannot draw her mouth to the right, but there is no inequality on smiling. She can move her arm and leg in all directions, but without much strength. Dorsal extension of the right foot is not performed as well as that of the left, and extension and abduction of the fingers of the right hand is attended with a little tremor. There is no contracture or rigidity, but the limbs are a little smaller on the right. She always was right-handed, but since her illness she has learned to write with the left hand. The heart and lungs were normal on examination. The plantar reflex is diminished on the paralyzed side; the abdominal and epigastric reflexes are absent on both sides. The triceps reflex was alike on the two sides; the radial and ulnar were absent, the patellar was exaggerated, and rather more marked on the right side. There was no clonus or front tap contraction. Sensation was perfectly good. The muscles of the face and arms react alike on the two sides to a weak faradic

current. Dr. W. N. Bullard, who saw her previously at the Boston Dispensary, and kindly permitted me to make use of his notes, reports that the galvanic reactions were tested by Dr. J. A. Jeffries, who found that the extensor communis digitorum showed  $KaSZ > AnSZ$  with  $\frac{3}{10}$  Ma.; the right peronei muscles,  $KaSZ > AnSZ$   $\frac{6}{10}$  Ma.; the rectus femoris,  $KaSZ = AnSZ$ ; everywhere else  $KaSZ > KaOZ$  and  $AnSZ > AnOZ$ . The following measurements show the difference in size.

					RIGHT.	LEFT.
Circumference of arm,	5	inches	above	olecranon,	6 $\frac{1}{2}$ in.	7 $\frac{1}{2}$ in.
"	"	"	5	"	styloid process of radius,	6 $\frac{3}{8}$ "
"	"	"	1	"	"	4 $\frac{3}{8}$ "
"	"	"	5	"	upper border of patella,	12 $\frac{1}{2}$ "
"	"	"	"	"	"	12 $\frac{3}{8}$ "
"	"	"	"	"	"	10 $\frac{1}{2}$ "
"	"	"	"	"	"	10 $\frac{3}{8}$ "

The right ulna was as long as the left, the semicircumferences of the chest were the same, and the legs were of the same length. As the paralysis is not of long duration, and as the child is already pretty well grown, we should not expect much of an arrest of growth in the bones as yet, if at all.

OBSERVATION V.—*Cephalalgia, probably of syphilitic origin. Right hemiplegia. Arrest of growth on right side. No contracture, but exaggeration of tendon reflexes.*

Michael R., 21, S., Plumber. Came to the out-patient department of the Boston City Hospital complaining of severe headache, weakness, and malaise. He seemed so ill that no careful examination was made. He stated then that his headache had lasted for five or six weeks, that he had used alcohol to excess, and that he had never had syphilis. For a month or six weeks he had noticed that his face was drawn a little to the left, and that his right side was weaker than the left. On stripping him there was found to be a distinct right hemiparesis, and a marked difference in the size of the limbs on the two sides of the body. Dr. Gleason, who was acting as externe, reported that the muscles of the arms reacted alike on the two sides to a mild faradic current. I am obliged to quote my own observations from memory, but I am indebted to Dr. C. F. Folsom, to whose service he was admitted, for kindly furnishing me a copy of the records of his case while in the hospital, as follows:

"Admitted June 15th, 1887. Family history good. Left-sided pleurisy two years ago. No other sickness. No syphilis. Drinks to excess. Not feeling well for two weeks, general weakness, frontal headache, loss of appetite. Bowels constipated. No abdominal or febrile symptoms. Last two days vomited three times. About six months ago noticed right hand smaller than left. No pain in right arm or leg, but they are not as strong as formerly; finds it difficult to use right hand in fine work. Eyesight and eyes all right.

"Well developed and nourished. Tongue moist, with a white coat, protruded a little to the right. Pupils normal. Left corner of

mouth drawn up a little. More contraction of left muscles than right when he knits his brows. Right grasp weaker than left. Heart's apex in or just outside of mamillary line. Prolonged first sound at apex; action a little uneven. Lungs and abdomen negative. Both knee-jerks exaggerated, especially right. Considerable tremor of leg when lightly tapped. Ankle clonus. Wrist clonus a little more marked right than left. On walking, right leg seems stiff, and foot occasionally drags. Stands well with feet together and eyes shut. No tenderness of shins. No neuritis or œdema. Patient right-handed. Left upper arm 2.5 cm. larger than right; forearm 1 cm. larger; hand at the metacarpo-phalangeal joints 2 cm. larger; right arm to end of middle finger 1 cm. shorter.

"June 23d.—Has had two or three attacks of extremely severe headache, relieved by morphine, and diminished by iodide of potassium, gr. xl., t. i. d. Now slight headache. Some dizziness and nervousness. Facial paralysis seems to be increasing. Up and about daily.

"June 28th.—Discharged at own request, against advice."

In spite of the patient's statement, it did not seem possible either to Dr. Folsom or myself that any recent lesion could cause such an atrophy of the right side, involving the bones; and furthermore, the benefit derived from iodide made us strongly suspect some syphilitic taint. It seemed to us not unlikely that he had had a hemiplegia in childhood, from which he had recovered, but that it had caused an arrest of growth on that side, and possibly an exaggeration of the tendon reflexes. This had passed unnoticed until recently, when a syphilitic process (endarteritis?) had developed at the site of the old lesion, as it is prone to do, causing weakness of the right side, and perhaps facial paralysis, and, with its attendant symptoms of headache and malaise, had called his attention to his right side, which he discovered for the first time to be smaller than the left. It is a matter of common experience that symptoms that do not cause discomfort are often overlooked, and, in a patient whose mental powers were as untrained as this man's, such a hypothesis is by no means improbable.

A glance at the illustrations to this article—notably those of Observations I. and III.—or at the patients themselves, would lead one very naturally to make a diagnosis of an old anterior poliomyelitis; but more careful examination will show that in all these cases the lesion is without doubt cerebral. The hemiplegic distribution of the paralysis, the implication of the facial muscles, the exaggerated reflexes, the spastic contracture seen chiefly in the arm, the normal electrical reactions, the presence of convulsions, all



point to a lesion in the brain and not in the anterior cornua of the cord. It is a cerebral infantile paralysis and not a spinal. In spinal paralysis hemiplegia is rare and the face is very rarely if ever involved; the tendon reflexes are absent or at least diminished, convulsions, except in the initial stage, are never present, and the characteristic reaction of degeneration, or the absence of faradic reaction or of both faradic and galvanic reactions, is to be expected.

There is one point of distinction in the cases reported which seems to me of decided importance, and that is the existence of epileptiform attacks after any symptoms of active disease have passed away. This I believe to be of great value in diagnosis as a means of differentiation between a lesion involving the cortex and a lesion farther down in the brain, deep down in the centrum ovale or in the region of the basal ganglia. In the first two cases I believe that the lesion is cortical; in the third and fourth that it is central; the fifth I imagine is central, but the impossibility of getting any definite history from the patient renders it useless to attempt to draw any conclusions from it, and I shall therefore leave it out of account.

Considering first the cases where epileptiform attacks were absent (Obs. III. and IV.), in Obs. III. the lesion has destroyed the left motor tract somewhere between the cortex and the pons, probably in the internal capsule, and has been followed by a secondary degeneration of the right crossed pyramidal tract in the cord. In Obs. IV. there is little or no secondary degeneration, and the lesion is probably not one which destroys the tract, but one which causes a break in the nerve conduction by pressure upon it, and it very likely lies in the lenticular nucleus or the corpus striatum.

The pathology of these cases is not without interest, although it has been less discussed, owing to the much greater interest aroused of late in the study of the pathology of cortical lesions in children, of which I shall speak later. The lesions in or about the basal ganglia giving rise to hemiplegia are the same as those in adults—abscess, tumor, embolism, thrombosis, and hemorrhage. That in

Obs. III. or IV. there is any progressive trouble, such as abscess or tumor, seems extremely improbable from the course of the cases, and the absence of any special symptoms, such as headache, vertigo, or optic neuritis.

At the last meeting of the American Neurological Association Dr. Sachs (61), in a paper of which only an abstract has yet appeared, called attention to the occurrence of intracerebral hemorrhage in the young, and laid especial stress on the absence of epileptiform attacks as a diagnostic point between intracerebral and meningeal hemorrhage. That intracerebral hemorrhage does occur in children has been repeatedly proven by autopsy, although most writers on children's diseases claim that it is extremely rare. Dr. Sachs stated that, according to certain observations made by Osler, miliary aneurisms occur in children; and furthermore, that Recklinghausen claims that fatty degeneration of the cerebral arteries, permitting transudation of blood, was not infrequent. In the discussion that followed Dr. Sachs' paper, Dr. Zenner called attention to the fact that there may be an embolism from endocarditis when no adventitious murmur is to be heard in the heart. The diagnosis between embolism and hemorrhage is so difficult as often to be impossible, and although in these two cases I am inclined to believe that there has been a hemorrhage, I do not feel that such a diagnosis could be confirmed without an autopsy.

Returning now to Obs. I. and II., in which epileptiform attacks were present, and in which the lesion was thought to be cortical, we enter upon a question in pathology which of late has been widely discussed. The discussion of the character of the lesion in Obs. II. need not detain us long. Evidently of traumatic origin, there has probably been a hemorrhage, either in the meninges or involving the cortex itself, yet not severe or extensive enough to destroy its functions. The absence of severe headache, vertigo, and other general symptoms makes the diagnosis of any other morbid process, such as meningitis or tumor less probable.

In Obs. I., however, the case is different. Here we have

a morbid process coming on spontaneously, involving the cortical gray matter of the left central convolutions, most markedly in the centre for the hand, but involving also the centres for the face, arm, and leg, and the motor centre for speech. From the cortex there has been a descending degeneration of the fibres of the pyramidal tract down into the cord. This is one of the cases to which Strümpell (70, 71) has given the name of acute poli-encephalitis, regarding them as similar in respect to their morbid processes to the cases of acute anterior poliomyelitis.

The disease attacks children in the early years of life, especially under six years of age, and comes on either during a period of perfect health or especially after some acute disease, particularly the exanthemata. It may begin with fever, delirium, vomiting, and diarrhœa, or, very commonly, with convulsions, and Strümpell suggests that poli-encephalitis may be the cause of death in those cases where a child dies suddenly with acute cerebral symptoms, such as convulsions. On recovering from this initial stage the child is found to be paralyzed, usually hemiplegia, the arm, leg, and lower part of the face being generally affected, although strabismus is sometimes seen. The arm is generally more helpless than the leg, as in the hemiplegia of adults. Contracture develops as in adults, and there is also a pronounced arrest of growth in the paralyzed limbs, involving the bones as well as the muscles. There may be a slight quantitative diminution of electrical excitability in the paralyzed muscles, although this is rare, but there are never any quantitative changes. Sensation is normal. The tendon reflexes are exaggerated on the paralyzed side and sometimes on both sides; but the cutaneous reflexes are absent or diminished. In right hemiplegia there may be disturbances of speech. Epileptiform attacks generally persist, and in many cases there are other motor disturbances—chorea, athetosis, ataxia, or associated movements. The child is very apt to be imbecile or idiotic. In the cases that have come to autopsy, porencephalia—a loss of substance or atrophy of the convolutions—has been observed, and many observers have

found traces of old inflammatory disturbance, spider and fatty granular cells. Strümpell, moreover, is inclined to regard this disease, as well as acute anterior poliomyelitis, as of infectious origin—a hypothesis which has received a remarkable support from a case cited by Möbius (48). Two children in the same family were attacked within twenty-four hours of each other with fever, loss of appetite, and restlessness. One child, a girl, in a day or two developed a typical anterior poliomyelitis, and her brother an equally typical spastic hemiplegia, with post-paralytic chorea.

Strümpell (70) closes his paper by saying that we must bear in mind that the hemiplegia of children may be due to other causes of which I have already spoken. Bernhardt (5) adds that if, "either suddenly while in apparently perfect health, or in the beginning, or during the course of convalescence from an acute febrile disease (especially the exanthemata), we notice in children in the first years of life a sudden outburst of unilateral convulsions, lasting for hours and followed by hemiplegia with the peculiar symptoms described (contractures, hemichorea, hemiathe-tosis, associated movements, etc.); and if, beside these symptoms, the psychical anomalies and especially epilepsy persist, we are justified, in my opinion, in recognizing this special form of cerebral infantile paralysis, this spastic cerebral infantile hemiplegia, as a more or less distinct *clinical type*."

So far most observers substantially agree. They also admit that the lesion of this "distinct clinical type" is situated in the motor region of the cortex, but the nature of the morbid process is still a matter of dispute. In the majority of cases the result of the process is porencephalia—a loss of substance in the brain involving the convolutions, or a cicatricial thickening and atrophy of the cortex. Whatever the primary process may be, it leads ultimately to this. Dr. Sarah J. McNutt (44), in a paper read before the American Neurological Association three years ago, tabulated thirty-four autopsies, of which thirty-two showed wasting or porencephalic defects in the cortex. I have

collected twelve other cases, in nine of which a similar condition was found [Bernhardt (5), Binswanger (7), Jendrassik and Marie [two] (33), Kast [two] (36), Mordet [50], Sander (62), Warner and Beach (73)]. One case [Seeligmüller (63)] revealed meningitis with softening of the cortex; in another [Beach (3)], the parietal convolutions were firmer and "coarser," *i. e.*, less well developed, and the microscope showed a great increase in the number of vessels, with distention of the vessels, and extensive infiltration with leucocytes, especially in the perivascular spaces. In the last case [Fraser (21)] there was a hypertrophy of the parietal lobe on the side opposite the paralysis, but the microscope revealed a defective development of cells.

The cause of this change in the cortex is still uncertain. In some cases where the trouble was congenital, Cazauvieilh (14) claimed that there was a defective development of the brain due to some intra-uterine disturbance. Little (41) attributed the trouble in his cases to injury of the brain received at birth. Strümpell's theory of an inflammatory process involving the cells of the gray matter of the cortex has already been referred to, and it is supported by Eichhorst (18). Kundrat (37) believes that porencephalia may be either congenital—a defect of development—or acquired. The acquired form follows the distribution of the arteries and is often of vascular origin, arising from an anæmia without arterial lesion, depending upon vascular weakness. This anæmia may be consecutive to pressure during labor. Porencephalia, he states, is a loss of substance from some destructive process in the brain, arising from hemorrhage, thrombosis, embolism, or anæmia without arterial lesion. Wood (76) and Bastian (2) assert that spastic infantile hemiplegia is due to cortical hemorrhage, but they do not say whether the hemorrhage may give rise to porencephalia; Ross (59), however, claims that it does. Jendrassik and Marie (33), however, could find no traces of hemorrhage or softening as a starting point for the sclerosis. In their two cases they found the perivascular lymph spaces greatly dilated, containing spider and fatty granular cells. This they regard

as the primary process with a secondary diffuse process leading to sclerosis and atrophy following it, the exact nature of the change being still in doubt. Henoeh (29) finds an interstitial growth of the neuroglia—a sclerotic process—with fatty granular cells and corpora amylacea. In this connection it may be said that Jastrowitz (32) does not regard the mere presence of fatty granular cells as proof of an inflammatory process, but only their aggregation into foci. The development of the medullary sheaths in the foetal brain is normally attended with the formation of fatty granular cells. Steffen (69) attributes the trouble, perhaps, to a meningitis. Gowers (24) maintains that these cases of hemiplegia are due to a venous thrombosis. This view is corroborated by a case reported by Heubner (30), where, in addition to a genuine porencephalia, a thrombus was found in the right artery of the fissure of Sylvius. The patient also had endocarditis. Lambl (38), who examined a case of congenital brain defect, gives as the causes of porencephalia congenital defect, aplasia from syphilitic endarteritis or embolism, loss of blood-supply, interstitial encephalitis, a fatty degenerative process, Kundrat's anæmia without arterial lesion, and perhaps hydrocephalus.

The latest contribution to our knowledge of the pathology of the affection has been made by Kast (36), who had an opportunity to examine two cases at an earlier stage in the disease than any that have been reported. He agrees with Bernhardt in thinking the lesion is not a pure poli-encephalitis, but also a chronic leuko-encephalitis, a diffuse sclerosis of the gray and white matter of the cerebrum. He failed to see the initial stage of perivascular changes described by Jendrassik and Marie, although he considered it impossible to decide from his data whether vascular changes were or were not the primary trouble; nor did he find Gowers' thrombosis of the veins. He believes it impossible at present to decide as to the primary change, owing to the lack of examinations of cases in the initial stages.

At present, therefore, it seems safe to say that probably several processes may give rise to porencephalia—hemor-

rhage, embolism or thrombosis, congenital defect or acute encephalitis, which may be analogous to acute anterior poliomyelitis and may possibly be infectious.

The prognosis must always be guarded. In Obs. I. and II., I have thus far been fortunate in holding the convulsions in check, but they often persist. Mild cases of paralysis, as in Obs. IV. and V. may make a tolerable recovery by the aid of tonics, cod-liver oil, massage, and electricity; but when there is pronounced secondary degeneration, as in Obs. I. and III., the prospect of recovery is of course hopeless. The patient may live to a fair age, although Henoch (29) claims that few outlive their twentieth year, but it must be remembered that they never have a sound brain, and thus must fall behind in life's race. Idiocy and imbecility are common sequelæ, although not present in the cases reported, and Mercklin (46) thinks that in those whose minds are not at first affected there is a lessened resistance and an increased vulnerability of the brain, so that later in life various psychoses may develop. Persistent and patient work, however, in the way of massage and gymnastics, may produce some gain even in bad cases.

Concerning two of the symptoms, I wish to speak further. Although four of these five cases were cases of right hemiplegia, the disturbances of speech were insignificant. In some cases aphasia may persist, so that the patient may remain mute for life, although Bernhardt (5) questions whether congenital aphasia, unattended with idiocy or deafness, ever exists. Clarus (17) believes that where there has been a destruction of the speech centres the prognosis is bad, but with slight and transitory disturbances of them it is good. Bernhardt thinks that aphasia in children is generally temporary, and usually of the motor type. In children, the right hemisphere may become educated as a speech centre, and thus acquire the power of speech after the centres in the left hemisphere have been destroyed. In support of this theory, he cites an observation by Kahler (35), where a woman had right hemiplegia from congenital atrophy of the convolutions,

including the left third frontal, with perfect power of speech. At the age of thirty-five she had apoplexy, causing left hemiplegia, from which she died. After her attack she was completely aphasic. At the autopsy a fresh lesion was found in the right hemisphere.

The other symptom to which I wish to call attention is the very marked arrest of growth in the paralyzed limbs. Seeligmüller (63) and Henoch (29) regard it as merely an atrophy from disuse, and assert that it is never so great as in anterior poliomyelitis. Such an assertion is untenable, for in some of these cases the atrophy is as pronounced as in poliomyelitis, and, as Förster (20) says, no atrophy from disuse could affect the growth of the bones. In certain cases of old hemiplegia in adults, Charcot,<sup>1</sup> Pitres,<sup>2</sup> and Brissaud<sup>3</sup> have found that, when there had been atrophy of the paralyzed limbs, the secondary degeneration had extended from the lateral columns to the motor cells in the anterior cornua, which they found atrophied. In some of these cases, however (Obs. II. and IV.), the signs of secondary degeneration in the lateral tract are very slight. In these cases, as in those of Ranke (54) and Seeligmüller (63), the electrical reactions were normal; Steffen (69), however, has noted a quantitative diminution, and Henoch (29) says that when the muscle is entirely wasted they disappear. I have not found this diminution in the hemiplegia of childhood, but I have seen it in adults, and in one case I have seen AnSZ > KaSZ which is, as far as my knowledge goes, unique.

In the cases reported, the various disturbances of motion—chorea, athetosis, ataxia, and associated movements—so often noted by other observers, were absent, except, perhaps, in the earlier course of Obs. IV. In the

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<sup>1</sup> Charcot, "Leçons sur les maladies du système nerveux." I., 55, Paris, 1872.

<sup>2</sup> Pitres, "Note sur un cas d'atrophie musculaire consécutive à une sclérose latérale secondaire de la moelle épinière." Arch. de physiol. norm. et path., p. 567, 1876.

<sup>3</sup> Brissaud, "De l'atrophie musculaire dans l'hémiplégie." Rev. mensuelle de méd. et de chir., p. 613, 1879.



other cases of cerebral infantile paralysis which I have seen, however, such disturbances held a prominent place in the picture of the disease; and at some future time I shall hope to present a study of them.

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72. Taylor. "A Case of infantile Hemiplegia, with unusual reflex Phenomena." Trans. of the Clin. Soc., XVI., 243, 1883.

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75. Wolfenden. "On cerebral infantile Paralysis." Practitioner, Sept. 1886.

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<sup>1</sup> See also this Journal and number, p. 503, 1887.

## Clinical Cases.

### "INTRACEREBRAL HEMORRHAGE IN THE YOUNG."<sup>1</sup>

By B. SACHS, M.D.

Within the last few years, the pathology of the infant's brain has attracted unusual attention. As a result of closer study, we have learned to differentiate between a number of forms of paralysis which were at one time included under the comprehensive term of 'infantile paralysis.' In this connection, I need merely remind you of our increased knowledge on poliomyelitis to be ascribed mainly to the careful researches of Kundrat, Henoch, Strümpell, Bernhardt, and in no mean degree to an excellent paper by Dr. McNutt. With poliomyelitis I am in no wise concerned in this paper. Leaving aside paralyzes to be ascribed to this condition, I am safe in saying that suddenly developed paralyzes, and hemiplegiæ in particular, have been too frequently attributed to meningeal hemorrhage. No doubt that in most, and in traumatic cases particularly, an effusion of blood into the meninges or over the cortex is the cause of the paralysis. It is my own conviction, however, that intracerebral hemorrhage is more frequent in the young than it is generally supposed to be. If so, it will be important to be able to make a differential diagnosis between these two forms of intracranial hemorrhage at a very early stage of the trouble, or rather between hemiplegia due to meningeal, and hemiplegia due to intracerebral, hemorrhage. If for no other reason, it will be important in order to give a correct prognosis.

I have said that I believed intracerebral hemorrhage to be more frequent in the young than it was generally considered to be. Withal, the condition is rare enough to merit full discussion, and I have therefore no hesitation in calling your attention to a few cases of this type.

I wish to anticipate criticism of my remarks by confessing that I have no autopsy to prove my diagnosis, and that I may be guilty of what Dr. Seguin once styled 'speculative pathology.' But

<sup>1</sup> This paper, although read a year ago at the meeting of the Am. Neurol. Association, has not hitherto been published, as the author wished to confirm his views by autopsies. In view of the appearance of Dr. Knapp's paper, the author has determined to publish these "clinical cases." Case I. has since died of malignant diphtheria, but unfortunately no post-mortem examination was granted.

there are conditions which rarely lead to the post-mortem table and yet are of commanding interest. Moreover, in the present state of cerebral pathology we are, I claim, fully able to infer pathological appearances from clinical data. The clinical data, and the inferences drawn therefrom, must be beyond dispute. You will find a close resemblance between the histories here presented to you and that of an ordinary adult apoplexy. This striking analogy may be taken as one proof of the identity of the pathological processes. The details of my first case are as follows:

CASE I.—One of three children; good family history; no syphilis.

The patient, M. M., is a healthy-looking boy two and a half years old. Up to the time of present illness in October, 1885, boy had had no other diseases but chicken-pox, eczema, and one and a half years previously, inflammation of the lungs. No history of fall. October 24th I was called to see the child and was told that it had been restless the night before, that it had vomited once, and seemed to have some difficulty in swallowing its food. Inspection showed that the tonsils were considerably swollen, the tongue slightly coated, but there was nothing of a diphtheritic character visible on or about the tonsils. The rectal temperature was  $101.4^{\circ}$ . During the next few days the tonsillar symptoms abated under the ordinary iron treatment. The temperature never exceeded  $102^{\circ}$ , and the pulse varied between 90 and 110. I saw the child on the morning of the 27th, found all its symptoms in abeyance, the child moved about freely, was feeling well and answered questions in its childish way, played with its toys, and did not seem in need of further medical attendance.

Early the following morning, the father called upon me in great distress, saying that his child could not speak and that it was not able to move its right side. My first fear was that the throat trouble had perhaps been of a more serious character, and that the child was now afflicted with a form of diphtheritic paralysis; but on seeing the patient I rid myself very quickly of this notion, for to my knowledge diphtheritic paralysis is never of the hemiplegic order.

At the patient's house I learned the following: On the day previous, soon after I had left the child, the latter seemed to grow very quiet; the nurse was struck by the fact that the child had given up calling her name dozens of times in succession as had been its habit; she did not remember having heard a single word pass the child's lips since that morning. No further attention was paid to the matter, as the mother and nurse thought the child sullen and nothing more. The nurse claims that the child used its arms perfectly well during the day, that it climbed of its own accord from one bed into another about 8 o'clock in the evening. At about 10 o'clock the mother noticed that the child moved its left arm and not its right, and the nurse noticed during the night that the child embraced her with its left hand only, that it lay on

its right side and did not seem able to change its position; the nurse turned the child several times during the night. Loss of consciousness was not observed at any time, although it is very evident that some paralysis had set in before the child fell asleep. The child was watched continuously day and night, and no convulsive movements of any sort, universal or partial, were observed.

On examining the child, I was greatly surprised to find typical right hemiplegia with aphasia—if you will allow the bull—adult hemiplegia in a child two and a half years old. I found that while the child seemed to understand all that was said to it, it could not be made to utter a single word. It moved its lips as though it wished to speak but could not. I thought, as I was about to leave the room, that it said good-by; but in this I may have been deceived, for the other persons in the room did not hear it, and certain it is that during the remainder of the day it did not utter a single word.

The right arm and right leg were markedly paretic, not absolutely paralyzed. It could move its hand a little, both spontaneously and when asked to do so; when lifted, the arm did not fall entirely "dead" upon the bed. The child was placed upon its legs for an instant, when it was evident that the right foot was dragged and that it made no independent movements. When the child was put to bed, I found that the right leg was slowly withdrawn after tickling the sole, but not after pinching the leg. There was slight and facial paresis. The knee-jerks could be elicited on both sides. Sensation normal in every respect, ocular movements were perfect; pupils equal and contracted promptly; water and fæces were passed voluntarily. The symptoms remained unchanged during the day, except that in the evening the child seemed less bright than in the morning. It did everything, however, which it was asked to do, showed its tongue, took its food, and so on.

Evening temperature in rectum  $100^{\circ}$ , pulse  $102^{\circ}$ . Heart sounds normal, optic discs ditto, urine free from albumin and sugar. No vomiting at any time.

During the next four days the condition remained practically unchanged. The child did not utter a word, but could be made to laugh when his little brother spoke to him. On the fourth day, there was a slight improvement in the movement of the leg, but none in those of the arm. The treatment consisted at first in small doses of sodium iodide, and ergot. This was soon abandoned for inunctions of the oleate of mercury. Absolute rest, ice to head and neck were the only other remedial measures employed.

On the fifth day the improvement was more decided; the child said "papa" when its father came near the bed, and spoke the end words of some little rhymes that it was accustomed to repeat when well. The right foot could be drawn up some distance, under the influence of a stronger incentive (holding its toe back);

the arm has not perceptibly improved; child laughs heartily, and when doing so the right facial paresis is more evident than when the face is at rest; it does not attempt to blow at watch; plantar reflexes equal; knee joints ditto. Sensation continued normal.

The further history of the case need not be given in full. The child recovered as adult hemiplegics do; first the use of the leg and then of the arm. On the thirteenth day after initial symptoms was able to stand on its legs unsupported. From the eighteenth day on, it began using its hand, scribbling all day long. From the thirteenth day on, began to speak in sentences. After three weeks all symptoms had disappeared, and during the past eight months the child has exhibited no other cerebral symptoms; but it has at present the whooping cough, the very trouble I feared most. Thus far it has done well and has escaped all further cerebral trouble.

On the third day, I made the diagnosis of intracerebral hemorrhage, probably hemorrhage into lenticular nucleus; prognosis was favorable from the outset.

The earlier history of the case I gave in great detail, in order to put the diagnosis upon a firm basis. The resemblance to ordinary adult apoplexy is so great that we might argue about the symptoms by analogy only; but I prefer to examine them in unbiased fashion. Two facts stand out most prominently; these are, no loss of consciousness and entire absence of convulsions. How do these symptoms bear upon the differential diagnosis; first, as regards the immediate cause of the apoplexy (using that word in its broadest sense), and secondly, as regards the differential diagnosis between meningeal and cerebral hemorrhages? Was the hemiplegia due to thrombosis, embolism, or hemorrhage? There are no facts which argue in favor of thrombosis. As for embolism, there is little in support of that possibility. There is no cardiac mischief, no history of rheumatism, and the mode of onset was slow, not sudden. If the history be correct, as given above, the child felt ill at ease during an entire day before the attack developed. The aphasia was developed first, the paralysis of arm and leg several hours later. There is but one statement, made by no less an authority than Nothnagel, which might lead us to give a different interpretation to these facts. In the article on cerebral hemorrhage in Ziemssen's large work, Nothnagel, discussing the differential diagnosis between embolism and hemorrhage, declared that if hemiplegia be recovered from within a few days this would argue rather against hemorrhage and in favor of embolism. This statement is misleading, for we must not forget that there are direct and indirect lesion symptoms—a fact which Nothnagel in later years helped to prove. Hemorrhage into the lenticular nucleus is a very different occurrence from hemorrhage into the internal capsule: in the one case the hemorrhage is adjacent to, in the other it immediately involves the greater part of the motor tract; in the one case it



might suspend function temporarily, in the other it would interfere with motor functions for a long period of time, according to the amount of damage originally done, the power of recovery of the parts involved, or the power of other parts to assume the function of those destroyed. It is well known, also, that embolism is far more frequently accompanied by loss of consciousness than hemorrhage. The plugging of even a very small cerebral artery is almost invariably followed by loss of consciousness. In this case, not the amount of injury done, but the suddenness with which it is inflicted seems to be the more important factor. While there is little or no reason to suppose that the hemiplegia is due to embolism, there is everything in favor of hemorrhage except the age of the patient.

The mode of onset was such as we should expect from slow and small hemorrhage. There was no loss of consciousness, and there was prompt recovery in the course of a few weeks. What the conditions are causing such hemorrhage we shall discuss shortly.

But might this not after all have been a case of meningeal hemorrhage; can we decide the question definitely? I repeat the close clinical analogy between this and adult cases would justify us in supposing the pathological processes to be similar, and in adults we know this form of apoplexy to be due almost invariably to intracerebral hemorrhage. Does the preservation of consciousness during the onset of the attack or the absence of convulsions help in making a differential diagnosis between meningeal and intracerebral hemorrhage? Loss of consciousness is an extremely variable symptom; it seems to depend rather upon the quantity of blood effused than upon the area involved. Not so with convulsions. A convulsion, if it is anything, is a cortical affair, the result of cortical irritation; small hemorrhage over or near the cortex will be apt to bring on convulsions, and this may be confined strictly to the motor portions of one-half of the cortex. Convulsions are the invariable accompaniment of meningeal hemorrhage. An examination of a large number of cases of meningeal hemorrhage in dispensary practice brings out this fact very distinctly.

Very large intracerebral hemorrhage might possibly produce convulsions in children, but the absence of such convulsions argues, to my mind, strongly in favor of intracerebral hemorrhage and against meningeal hemorrhage. The symptoms of both may resemble each other in every other respect; hence the importance of paying special attention to this point. The prognosis in intracerebral hemorrhage is certainly far more favorable, and in my own case the absence of convulsions enabled me on the third day after the attack to prognosticate complete recovery.

In the early stages of acute polioencephalitis the symptom may in some respects resemble those discussed here. That is, the polioencephalitis may give rise to hemiplegic symptoms, but in my

case the preservation of consciousness, the lack of convulsions and the prompt recovery above all things, argue against the presence of polioencephalitis.

The rarity of intracerebral hemorrhage in the young, in fact, in persons under forty, is acknowledged by all, and in the case which I wish to refer to now the diagnosis was considered uncertain by several neurologists, and yet this patient was nearly seventeen years older than my first case.

CASE II. I had the privilege of seeing in private practice only a short time ago. The patient is a young man about nineteen years of age, with good family history, of excellent habits, and without hereditary or acquired syphilis. He is by no means frail in appearance, though rather undersized, and his father states that the boy stopped growing suddenly. About a year and one-half ago he was preparing very earnestly for his college examinations, when he was seized with a slight left hemiplegia. The onset was gradual, there were neither coma nor convulsions; and after three weeks, during which the symptoms had receded typically, he had completely recovered. He went along very well for exactly one year when one day, as he was paying a visit, he felt numbness coming on as it did in the previous attack. He had courage enough to start for home; when he arrived there he began to vomit, became comatose (one and one-half hours after initial numbness) and remained in this state of coma for eighteen hours; no convulsions. He had complete left hemiplegia, but no aphasia although he was congenitally left-handed; right-handed by education.

The paralysis remained unchanged for a fortnight. Recovery began with leg; now he has full use of his lower extremities, though he drags his left foot in characteristic fashion. He has exaggerated knee-jerks on both sides, and very marked double ankle clonus; his left hand is still paretic and contractured, no cardiac or renal trouble. The diagnosis reads intracerebral hemorrhage, involving destruction of a large part of the internal capsule. In this case, mental overwork is considered the direct exciting cause; in the first case, no direct cause could be made out. The child had no acute infectious or exhausting disease, and had not at that time the whooping cough—causes to which intracerebral hemorrhage in the young has been ascribed by writers on the subject. I cite the second case to show the analogous symptoms in the two. In both hemorrhage occurred in persons far below the usual age limit; in both syphilitic changes can be safely excluded. Convulsions were absent in both, while in the one case coma supervened during one of the two attacks.

The second case proves the danger of this tendency to hemorrhage when exhibited in very young persons—the danger of repeated attacks. In this regard the prognosis must be held to be unfavorable.

And now as to the pathology of the two cases. This is at once

the most interesting and the least satisfactory aspect of the entire subject. The analogy with adult cases would lead us to suppose that hemorrhages in these cases were due to the rupture of miliary aneurisms of the middle cerebral artery. The probability is very strong, but the reports of post-mortem examinations which showed intracranial (including meningeal) hemorrhage in young persons to be due to the rupture of small aneurisms are exceedingly few. In his classical work on the "Pathology of the Circulation and Nutrition," Recklinghausen states (p. 84) that he has seen but a single fatal case of intracerebral hemorrhage in the young, and this was a case of chronic renal disease with hypertrophy of the heart. In this connection I may refer to a publication of Dr. Osler in the *Canada Medical and Surgical Journal* (1886), in which he reports the finding of an aneurism of a branch of the anterior cerebral in a boy six years of age; as Dr. Osler adds, to his knowledge the youngest case on record.<sup>1</sup> In view of this paucity of knowledge on this subject, we must stop to inquire whether hemorrhage may not be due to other conditions than those of aneurism? There is but one change in the blood-vessels of the young which is familiar to many pathologists, and that is a fatty degeneration of the walls of smaller blood-vessels, and again Recklinghausen suggests that there may be a simple transudation of blood through these pathologically altered blood-vessels. The pathology of the vascular system of the young is a subject of surpassing interest. It is almost virgin soil on which some of us should plough successfully.

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<sup>1</sup> Boy, aged 6, brought to hospital unconscious with feeble pulse, pale face, eye's and head turned to right, and left hemiplegia. Death in six hours. He had fallen from a hay loft three weeks before, but he recovered rapidly from the effects. There was meningeal hemorrhage at base and in longitudinal fissure. An aneurismal sac was found imbedded in the callosal-marginal fissure just where it turns vertically upwards. The rupture was on the meningeal surface, but hemorrhage had extended into contiguous portion of brain. The arteries were not atheromatous, presumably altogether normal, and the heart was healthy.

## Society Reports.

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### AMERICAN NEUROLOGICAL ASSOCIATION.

*Nineteenth Annual Report.*

*Wednesday, First Day, Morning Session.*

The American Neurological Association convened at the West End Hotel, Long Branch, N. J., July 20th, 1887, and was called to order by the President, Dr. CHARLES K. MILLS, of Philadelphia, who asked that the by-laws be suspended temporarily to receive a report from the Council. The request was granted.

#### REPORT OF THE COUNCIL.

Dr. GRAEME M. HAMMOND, of New York, Secretary, reported that the Council had unanimously recommended the election of of the following applicants for membership: Henry Hun, of Albany; Henry M. Lyman, of Chicago; Irvine C. Ross, of Washington; Theodore H. Kellogg, of New York; and Philip Coombs Knapp, of Boston.

They were elected by a ballot cast by the Secretary. The following

#### AMENDMENT TO THE CONSTITUTION

was then adopted. Article 3 of the Constitution shall be amended so as to read as follows:

“There shall be two sorts of members, namely, active members not exceeding at any one time one hundred in number” (instead of fifty).

On motion, the reading of the minutes of the last annual meeting, already published, was dispensed with.

#### MEMBERS PRESENT.

The following members were present during either the whole or a part of the meeting:

Drs. Charles K. Mills, of Philadelphia; Landon Carter Gray, of Brooklyn; Robert T. Edes, of Washington; F. T. Miles, of Baltimore; James Hendrie Lloyd, Francis X. Dercum, and Wharton Sinkler, of Philadelphia; J. J. Putnam, of Boston; Henry Hun, of Albany; Graeme M. Hammond, B. Sachs, E. C. Spitzka, Ralph L. Parsons, M. Allen Starr, R. W. Amidon, Theodore H. Kellogg, C. L. Dana, George W. Jacoby, E. D. Fisher, and Nathan E. Brill, of New York; and Isaac Ott, of Easton, Pa.

#### MEMBERS BY INVITATION.

Dr. J. Jastrow, of Johns Hopkins University, and Prof. Samuel Ayers, of Pittsburgh, Pa.

#### REPORT OF THE TREASURER.

Dr. Graeme M. Hammond, of New York, Secretary and Treasurer, presented the annual report, which, on motion, was accepted. There was a balance in the treasury of \$110.00.

After the transaction of the above preliminary business, the retiring President delivered the following address:

#### ADDRESS OF DR. C. K. MILLS.

At the meeting of this Association last year, an amendment to the Constitution, which I have no doubt will be acted upon favorably, was offered as follows: "The officers shall enter upon their duties immediately after the adjournment of the annual meeting at which they are elected." The offering of this amendment, and the favor with which it was received, shows that the Association is fully satisfied with one formal address by its President, and receives this by preference at the inauguration of his term of service. I will, therefore, detain you but a few minutes before making way for my distinguished colleague, the President elect. Otherwise I am sure you will all feel like saying, "Welcome the coming, speed the parting guest."

I may perhaps be permitted brief reference to some of the other amendments which were offered last year, and will come up for final adoption at one of the sessions of the present meeting. The first of these, offered by myself, was to substitute two Vice-Presidents, instead of one Vice-

President, in the list of officers of the Association. One reason for offering this amendment was to enable us to make a somewhat fuller and better distribution of officers, and thereby to improve, if only to a slight degree, the national character of the Association. If this amendment is carried, the President and each of the Vice-Presidents should, as a rule, be chosen from different States or different sections of the country. Such a rule in a society composed of comparatively few members could not be made absolute, but a following of it by tacit agreement, when practicable, will, I think, advance the interests of the Association.

Another amendment, offered at the last meeting, and which I trust will be adopted at this, is as follows:

"There shall be two sorts of members, namely, active members, not exceeding at any one time one hundred in number" (instead of fifty as at present). With reference to this amendment I can only reiterate, in advocating its adoption, the sentiments which I expressed in my Presidential address of last year, namely: "Our membership ought to be at least doubled by the addition of acceptable physicians from all parts of the country; for the American Neurological Association should be in fact as well as in name the great national representative body of those members of the American profession who are interested, either practically or theoretically, in neurology and psychiatry, and the branches of science which are especially correlated and auxiliary to these subjects."

A fourth amendment, offered by Dr. Jacoby, is to the effect that the officers shall be nominated by the Association on the second day of the meeting (instead of the first day). As the object of this amendment is to have this important function performed on that day of the meeting when the probability of a full attendance is the best, I have but little doubt that it will meet with unanimous approval.

Finally, as a matter of business which has continued to engage attention during my term of office, I will again refer briefly to the Congress of American Physicians and Sur-

geons. At the last meeting, it will be remembered that a resolution indorsing the propositions for the proposed congress was adopted, and a committee of conference was appointed as follows: Drs. L. C. Gray, of Brooklyn; J. Van Bibber, of Baltimore; W. Sinkler, of Philadelphia; E. C. Seguin, of New York; and Philip Zenner, of Cincinnati. Dr. Gray, chairman of this committee, will report to the Society as to its movements and action since our last meeting; and I will therefore only say that I trust the Association will promptly approve of the committee's report, and appoint a representative and alternate to the executive committee which is to make the final arrangements for the meeting of the Congress in September, 1888.

It will be remembered that the main portion of my address last year was devoted to a consideration of the subject of "Arrested and Aberrant Development of Fissures and Gyres." Several references were made in this address to Prof. Moritz Benedikt, of Vienna, and his work in connection with the subject of the brains of criminals. Since the last meeting of the Association, I have received a letter from Prof. Benedikt, and as in it he refers to my address and some criticisms of his own views (which he regards as not properly stating his position), and as he expresses a wish that I shall place him right with his colleagues in America, I shall take this opportunity of quoting from his letter; so that his remarks may be placed on record as part of the proceedings of our Association. The letter, with a few verbal changes and omissions not important to the main question discussed, is as follows:

"VIENNA, March 1st, 1886.

"MY DEAR SIR:—I thank you for sending me your interesting presidential address read at Long Branch. . . .

I see by your publication that my principal intention to excite the interest of the profession for these questions has succeeded in the United States. As I spoke the first time about the anatomical researches on brains of criminals at Paris, 1878, at the Congress, I concluded my speech with the words: 'I protest in advance against the conclu-

sions of the others, as you see, for example, from the pamphlet (one of a number sent to me by Dr. Benedikt) at the congress for criminal anthropology in Rome, I was obliged also to protest against the conclusions of our famous Italian collaborators.'

"Before all I must protest against the *legend* which exists that I have created a fixed anatomy of crime. If I had the opinion to have created a characteristic type of brain for crimes, I had entitled my book: 'Anatomy of Crime,' and not: 'Anatomical Studies on Brains of Criminals.'

"Now I know, with full security, that there exists an anthropology of a typical, abnormal, and deficient man, but not—in our time—of crime. I have found it true from the rich comparative study of heads of epileptics, of criminals, of the insane, and all those with diseases of the brain which are congenital or acquired in early youth.

"When you read my classification in 'Des Rapports existant entre la folie et la Criminalité,' you will see that I reckon under criminality a large number of individuals of normal organizations who become criminals through biographical circumstances, and who cannot have atypical structure of the brain, etc. Nevertheless, you make me (page 20 of your paper) the supposition, as if I had an absolutely fixed type of criminal brain. In opposition to my predecessors in anatomy of the brain, I had found that the principal fissures are not generally separated as those described.

"Au contraire, I found that in general there exist many conditions of the fissure, and I divided the brains in two types; the ones in which the fissures are more separated, and the others in which the fissures are more confluent, and I said only that the brains of criminals studied by me, *belong* to the second type. As you will see principally in the copy of the letter to Prof. Giacomi, I do not attribute a great importance to the confluence *pur et simple*, but only to those confluences which are very rare in normal brains, as, for example, the confluence of the Sylvian with the central fissure, of the perpendicular-occipital with the interparietal, of the calloso-marginal with the perpendicu-



lar-occipital, etc. In the same way I consider certain separations, as of the calcarine from the perpendicular-occipital as important.

"My dear colleague, you would oblige me very much if you would communicate in the way you find convenient, my protest against the named legend to our colleagues of America.

"I would not like to be identified with my 'followers and admirers.' When you read my reserves (page 157 of my book, the last ten lines), you will see that I defended myself in advance against the false conclusions and premature cry of triumph of my 'admirers.'

"It will interest you to hear that all young professors of jurisprudence in Germany are no more in opposition to me, and that also in Italy the priests agree more with me than with Lombroso and Ferri. When you will be so kind as to look another time in my book and to read my further publications, you will see that my opinions, views, and doctrines are quite other than cited in literature, and you have told me exactly (page 29 of your paper) how I was misunderstood, but you will concede more than 'partially.'

"In one publication, *Anzeiger der K. K. Gesellschaft der Aerzte*, No. 48, 1885, you will see that I have presented to that Society three Chinese brains. I have not yet published the description of them, but I will do so the next time, and then I will send you a copy.

"My dear colleague, I find my letter has become too long, and its bad English will not increase the pleasure of reading it.

"Accept the expression of his best feeling from yours,  
"F. BENEDIKT."

With these few remarks, I take pleasure in introducing my successor, Dr. Landon Carter Gray, of Brooklyn, N. Y.

The President, DR. LANDON CARTER GRAY, of Brooklyn, then delivered his Inaugural Address.

## ADDRESS OF THE PRESIDENT.

In thanking you for the honor which you have conferred upon me, I have deemed it best to remind you of certain facts pertaining to our Association and the branch of medicine in which we are all interested.

It was only in 1872 that the organization of neurological societies began in this country with the formation in that year of the New York Neurological Society. In 1875, The American Neurological Association had its being—only twelve short years ago. Three years ago, the Philadelphia Neurological Society was also organized; so that it has been entirely within the period of fifteen years that the neurological specialty has had, so to speak, official voice and expression. When we contrast ourselves with our European compeers, we find that we have done much more in this line than have they. Germany has only two neurological societies: one in Berlin, and the *Wander-versammlung der südwestdeutschen Neurologen und Irrenärzte*, unless we can say that the neurological section of the *Verein für innere Medicin* is an organization of that class. France has no neurological society by that name. England one year ago gave birth to the Neurological Society of London. Italy has a psychological, but none of a neurological character, in the broad sense of the word. Thus, whilst England and continental Europe, with their old civilization, their hoary universities, their teeming medical journals, have only three special neurological societies, one of them started within a year, this new country of ours has three, one of them fifteen years of age. This is certainly a startling contrast, and I think it might prove of some interest to us to search for the causes of the difference. I was very much struck with the remark made last fall by a distinguished English physician who was visiting this country, and who said that what impressed him most forcibly about America was the avidity with which a new idea was seized and practically carried out. I believe that this national aptitude or weakness, as you may choose to call it, is the main cause of the growth of specialism in our country. Talk about it as we may,

deprecate it as much as some of our friends do, there can be no doubt whatsoever that the tendency of the day is toward specialism. The modern growth of science has made this tendency inevitable. It would be physically impossible to crowd Ziemssen's Encyclopedia or Pepper's "System of Medicine" into the compass of the works of Galen, Hippocrates, or Celsus, and so the vast modern accumulations of facts that are being added to daily render it impossible for any man to master them in all departments of medicine, be he endowed with the mind of Bacon, or Shakespeare, or Goethe, or Napoleon; and even supposing that such a master mind were to be able to digest and assimilate the facts, he would still have before him the task of familiarizing himself with the specializing technique of each particular specialty, which an earnest, conscientious and industrious man may well congratulate himself upon mastering in a decade. I have but scanty sympathy, therefore, with the attacks that are made upon specialisms. One might as well attempt to dam Niagara with a teaspoon. I believe that the time of the critics would be much more profitable spent in calling attention to the abuses of specialism, and to the setting up as specialists of gentlemen who are insufficiently qualified in the general departments of medicine. Be this as it may, however, I do not see how any one can doubt the propriety of a specialty of nervous and mental diseases. When we consider the great complexity of the anatomy of the nervous system, the equal complexity of its physiology, and the still greater complexity of its diseases, and when we consider the marvellous activity of research in this department in the last twenty years, it would certainly seem as if it needed all the faculties of an intelligent man to keep himself abreast of the progress that has been made. We may fairly say that neurology has been made within the last quarter of a century. Before that time it was but a very inchoate science, and the art that went with the science was very meagre indeed. Just think alone of what a revolution has been effected in the doctrine of cortical localization! If any student, coming up for his examination in 1869, had

stated that muscular movements could be produced by electrization of the brain, he would have been thought too ignorant to be given a diploma. If a year later, after the experiments of Fritsch and Hitzig, toppling down with one dingy little article and one or two rude woodcuts all the venerated authority of Flourens, that same student had been examined on the same subject and had *not* stated that electrization of the brain would produce muscular movements, he would certainly have been in equally great danger. Look at the vast literature that has sprung up concerning this doctrine of cortical localization. Look at the wonderful experiments of Munk, Goltz, Ferrier, Luciani, Seppili, Nothnagel, Gudden, and others too numerous to mention. Look at the thousands of careful pathological observations that have been rained down upon this question from every quarter. Look at the great advance in our knowledge of the anatomy of the brain and spinal cord, gained by the destructive methods of Gudden, by the tearing methods of Meynert, embryological methods of Flechsig, and by careful microscopical observation; at the great advance and improvements in our microscopical methods and our microscopical instruments. Look at the advance which has been made in the differentiation of diseases, as in the demarcation of the different forms which have the anterior horns of the spinal cord as an anatomical substratum; the quick growth of our knowledge in regard to multiple neuritis, that form of disease which had remained mysterious in the East for centuries under the names of beri-beri and kak-ke, and which was touched into rapid and easy explanation hewn brought to the notice of the higher civilization of the West. Look at the growth of our pathological and clinical knowledge of syphilis of the nervous system. See how the nervous diseases arising from reflex action have come to be recognized as from urethral, ovarian, ocular, nasal, and laryngeal irritations. Contrast for a moment with twenty-five years ago our present knowledge of neurasthenia, lithemia, hypnotism, athetosis, lateral sclerosis, locomotor ataxia, brain tumors. See how our in-

sanities have been more accurately recognized and more humanely and medically treated ; how that most baneful of diseases, epilepsy, is controllable by the bromide treatment which we owe to Brown-Séquard ; how electricity has been changed from one of the most vague and unsatisfactory departments of medicine that ever attracted the eagerness of charlatans into one of the most precise and accurate methods of therapeutic and prognostic and diagnostic procedure ; how the growth of knowledge regarding cerebral localization is lending, in some respects, the most exact precision to cerebral surgery ; how hydrophobia is becoming a thing of the past, if Pasteur's claims may be trusted ; how the ophthalmoscope, by enabling us to utilize the peculiar disposition and vascular supply of a peripheral nerve, has made possible the diagnosis of what are sometimes otherwise undiagnosable diseases. In the face of these facts, a man may well stand with a serious countenance, even if he be disposed to give up his whole time and attention to the study of the nervous phenomena of which I have only spoken partially. It would be simply impossible for any one who proposes to remain a general practitioner to swallow and assimilate all this varied knowledge. Besides that, it has only been within a very recent time that there have been any facilities for the thorough acquisition of this specialistic knowledge in this country. Nervous and mental diseases have usually been honored only with a short and perfunctory course of lectures in the different medical colleges of the country. it has not been incumbent upon the medical student to take these lectures, he is not examined upon them, and unless he be endowed with a desire for knowledge which looks beyond the immediate exigencies of his graduation examination, he is not likely to pay much attention to them. Most of us, I think, who have been in practice ten years and upwards will look with envy upon the facilities for neurological study which are now extended to the young medical men in the shape of clinics, lectures, articles, and text-books. Ten years ago and over our knowledge was acquired in no such easy way. It had to be

gleaned from our own observation and our reading mainly of foreign journals and text-books. We have witnessed, as it were, the development of our specialty, and to keep pace with this development has been a much more difficult task, one that required a man to be much more on the alert than would have been the case if we could have turned to the well-digested and well-sifted text-books of the present day. It would seem as if the medical world at large were waking up to a realization of the great importance of a proper comprehension of nervous phenomena. I presume that most educated physicians would agree with me in believing that no man is a competent practitioner of medicine unless he is acquainted with the circulatory system of the human body. I venture also to presume that the time will come, and that before we men of middle age are dead, when educated physicians will also believe with me that no man is a competent practitioner unless he is thoroughly acquainted with the phenomena of the nervous system. The circulatory system got the start of the nervous system by a number of centuries; and very naturally, because its phenomena were easier to study; and that is the only reason why the profession appreciates its importance and overestimates it relatively to that of the nervous system.

I have made these remarks, gentlemen, not because I think they are either needful or perhaps particularly interesting to you as specialists, but because it has seemed to me that, outside of your own ranks, there is a lack of appreciation of the delicate and important work that is being done by neurologists, largely, I believe, because of the non-recognition of the various facts to which I have been making allusion.

I sincerely hope that this Association will become a member of the new National Congress of Physicians and Surgeons. I can see no reason why it should refuse to do so. The autonomy of this Association will not be disturbed in the slightest degree, as we are to hold our meetings every year separately, and only to take part every second year for a short time in the joint meetings of the new organiza-

tion. We shall have opportunities, that can only be offered by such a body, of meeting gentlemen who are distinguished in other departments of medicine; and if any good ever comes from the mental clash and stimulation of medical bodies, it certainly seems to me that a large amount of benefit should come to us all from the mental clash and stimulation that would be afforded by such a large cosmopolitan society as the new organization will be, for we shall see in it the picked men of every branch of our profession.

There is another subject to which I desire to call your earnest attention, and that is the amendment to Art. IV., third clause, of the by-laws, providing (in effect) that the president who is elected at this meeting and who presides at the meeting next year shall be put in charge of the arrangements for the meeting over which he presides. As it is now, I am sitting at the head of the table which has been furnished forth by the kind efforts of my distinguished predecessor, Dr. Mills; not that I have any objection, but that it is rather unjust to Dr. Mills, and leaves entirely too much work to him and too much play for me. I am at a loss to know how any such by-law could have been passed except by some oversight, and I earnestly recommend the adoption of the amendment.

DR. E. C. SPITZKA, of New York, then read a paper entitled

ON SOME POINTS REGARDING THERAPEUTICAL AND OTHER IN-  
JURIES OF THE BRAIN.

In the course of certain experimental inquiries undertaken between eighteen and three months ago, I made some observations of a surgical character which to me seemed novel, and to which no reference in the writings of other experimenters has been found by me. The experiments referred to varied from the excision of entire hemispheres, simple trephining, and the introduction of foreign substances into the arachnoid cavity to the injection of such substances into the brain tissue directly, into the ventricular cavities, and into the arachnoid sacs at the

base of the brain. Terebration or trephining necessarily preceded the introduction of hypodermic needles in the latter instances.

It was also my fortune to witness in one instance an extensive operative exposure of the human brain, and to make the post-mortem analysis in two cases where operations had been attempted or made in accordance with the modern teachings of cerebral surgery.

It is not necessary for me to state that under strict antizymotic precautions the amount of brain tissue that may be removed from animals without compromising life is practically unlimited as long as the brain axis is not injured in special provinces. Uniformly, I have been able to abuse this organ in dogs to an extent which some years ago would have appeared incredible, even without strict antisepsis; in a large number of cases, without any antiseptic precautions, and in a few under intentional violation of the principles of surgical cleanliness.

The results to which I would especially direct attention are those relating to the healing of wounds made by hypodermic needles and trocars. C—— N——, aged 11 years, suffered from symptoms which were interpreted by Dr. Cypert, his family attendant, as indicating a cerebral tumor. He was thrown down by a horse in the early part of the year, and certain of his symptoms became aggravated. He was taken to St. Luke's Hospital, and a suspicion having been expressed that he was suffering from a cerebral abscess, trephining at the seat of the injury was performed, and a hypodermic needle introduced three times in various directions. No fluid was obtained, the button of bone was replaced, the periosteum united, and the wound closed. No untoward result followed. The symptoms which to my mind, as well as to Dr. Cypert's, indicated a contralateral tumor progressed, and the diagnosis previously made was adhered to. He died comatose in May, three months after the operation. The autopsy was performed the same day.

On removing the calvarium, it was found that the dura bulged corresponding to the trephined spot. It was inti-



mately adherent at the meso-caudal third of the trephine circle, but altogether disconnected from the bone at the ecto-cephalic third. Its structure was entirely normal. On removing the dura, an exact reproduction of the outline of the button was found in the arachnoid. The latter was milky white and thickened in a circular area, the transition from the opaque to the clear arachnoid being very abrupt. At the ecto-cephalic third it appeared villous, and rose steeply above the general surface to a height of two millimetres. Corresponding to this rise, the underlying gyrus exhibited an anfractuosity.

The cerebral hemisphere, I may add parenthetically, which alone concerns us here, was entirely healthy aside from the slight traces left by the operation. The tumor was found where it had been located during life—in the opposite cerebral crus.

On searching for an indication of the punctures made, none could be discovered on the surface of the brain nor in the pia, arachnoid, and dura.

On carefully making thin sections of the region involved, three dark bluish lines were found extending vertically to the surface. One, the longest, measuring a centimetre in length, nearly reached the surface, and in the hardened specimen, a slight pucker was found to mark the point where it would have reached if it continued. The three showed the same composition—coagulated blood<sup>1</sup>; at their deeper ends there was a rusty-colored continuation, which microscopic examination showed to be due to the presence of a large number of blood-corpuscles on the otherwise normal cerebral tissue.

There were no spider cells, nor indications of any inflammatory disturbance whatever in the cortical or medullary tissue, nor did any of the nerve cells, as far as they could be identified, exhibit structural changes.

The blood-vessels were normal. I could discover no certain indications as to which, if any, vessel had been wounded.

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<sup>1</sup> Invading the corresponding temporal lobe, which had not been suspected. This case will be published in detail.

Undoubtedly the three delicate tubular semi-clots represented the entire extent of the permanent damage inflicted by the probe-punctures. So far as this case goes, their harmlessness is proven. In one other respect this case constitutes a remarkable confirmation of the soundness of recent surgical innovations. I refer to that of re-inserting the button of bone removed in trephining, and allowing it to reunite with the cranial vault from which it had been removed, as McEwen suggested and Weir has ably carried out.

It may be remembered that I described the thickened arachnoid, as well as the gyrus corresponding to the ectocephalic third of the trephine circle, as rising above the surface. Here also the periosteum had not united over the rim, while the dura was non-adherent and dull, exhibiting a doubly bridged solution of continuity  $1\frac{1}{2}$  centimetres in length.

Corresponding to this, the button of bone was raised above the surface, and had failed to contract union with the rest of the skull. But at the opposite point it had united in nearly its entire thickness from the dural aspect outwards, while the ditch cut by the trephine and the central depression were as complete on the outer side as if they had been freshly made, excepting for the union of the periosteum at the cauda-mesal arc. Not only was there a complete obliteration of the saw line on the dural side at this point, so that the inner contour of the calvarium was restored, but angular processes of newly-formed bone jutted up from the circumference, where they were rooted with a broad base, to unite with the button by their apices, thus constituting, as it were, a spurious sutura serrata. The button was slightly movable at the loose end, but only a pressure of several pounds would have severed the connections mentioned.<sup>1</sup>

From this it is evident that in young persons, even under well-discouraging circumstances, as wounding of

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<sup>1</sup> Before the suggestion to remove and preserve this interesting specimen by removing a fenestra from the skull was made, the scalp had already been closed.

the dura by the saw, partial hernia of the brain, and lifting of the button, the dura made successful efforts to agglutinate the removed button and restore the integrity of the calvarium.

In connection with a number of cases where trephining was practised, I have found the almost self-evident proposition illustrated—that the larger the opening the less is the probability of protrusion of the brain. In the case reported by Dr. Graeme Hammond, the square area of removed calvarium was certainly the largest on record among the cases where it was undertaken for medical purposes. Yet the extent of the protrusion, which is accurately marked in the alcoholic specimen, is not above a few millimetres, and this notwithstanding the pathological increase of intracerebral pressure.

Immediately under the cortex, in the region bordering on the white substance of the post-central gyrus, in a thin layer of tissue overlying the smaller cyst, also in the white substance ventrad of the larger cyst, I found small recent hemorrhages. It is probable that these resulted from the too sudden relief of external counterpressure. The danger of hemorrhage from this source should be borne in mind in all operations involving the removal of a large part of the cranial rolls.

The observations made with hypodermic and other needles on the brains of dogs relate to three points.

First, the formation of cerebral abscess at points remote from the point of injection of septic material.

In May last, a mongrel collie had fluid from a cerebral abscess, to the amount of a twentieth of a drop dissolved in bouillon, injected inwards and downwards; it did not reach the ventricle. On the autopsy, five days later, some injection of the pia was observed on both sides, most marked on the same side. A large yellow focus was found in the ectoventral part of the hemisphere in no connection with the channel of the needle. There was noticeable irritation around the latter, but no contiguous tissue-change connecting it with the abscess. Indeed, the ventricle intervened between injury and lesion.

I am able to exhibit almost the counterpart of this result in a specimen that has not yet been thoroughly dissected.

The second observation relates to the constancy of the channels made by exploring needles in brain tissue. We have already seen, from the human case cited, that practically hypodermic needles can be inserted with perfect impunity.

My experience with animals has been remarkably different in this respect, and would seem to indicate an enormous individual difference. On the one hand, coarse needles have been introduced, coarse substances injected, the animal killed after a month, and not the slightest trace of the channel or the point of penetration found. On the other hand, comparatively innocuous substances have been injected with fine needles, and the animal being killed after a year, the tract of the needle was found as fresh as if it had been made a few hours before.

It seemed to be indifferent how deeply the needle was inserted, whether it penetrated or passed the ventricles or reached the basilar arachnoid sacs. Experiments undertaken under apparently the same circumstances yielded different results in different animals. As a rule, it was in vigorous, large animals that the channel of the syringe was found obliterated, and in those with the opposite characteristics that it was found patent after a long period. It occurred to me that a hemorrhage might account for it. A remarkable exemplification of the former alternative was that of a mongrel dog of powerful, bony build, whom I intended to kill by inducing suppurative encephalitis. My cultures of the streptococcus were interrupted, and on the spur of the moment I used a material which had uniformly produced even more furibund lesions, namely, a drachm of New York street mud scraped from beneath the hoof of a horse in the stable where the dog in question was kept.

The skull being trephined, the needle of a hypodermic syringe was introduced, and the entire drachm of street mud previously triturated was injected, as it was sup-

posed, into the thalamus. The dog recovered from the rapidly performed operation without any untoward symptoms. For one month he showed no signs of any kind whatever. For two weeks following he became surly and snappish, and finally recovered, acting in every respect as healthy dogs do. About four months after the operation, he became afflicted with the "mange" and was killed. His brain, which was hardened in alcohol and sliced in a number of sections, about thirty to the inch, showed no signs of the entry point of the syringe, of its channel, nor of the carbonaceous mud that had been injected. In the arachnoid of the ponto-chiasmal lamina two large masses of dirt, held together in a transparent mucoid substance, were found. They were not firmly united to the arachnoid, for on incising the latter they escaped together.

A third mass of the same character was found in the arachnoid lamella extending on the basilar face of the oblongata.

The inability to discover the channel of the needle in this case is all the more remarkable, as the point of skull trephination could be easily identified. This is exceptional.

In two other cases where the needle was apparently introduced in the same direction, and simple emulsion of calf's cud in unsterilized bouillon had been injected in quantity not exceeding a drop, not only did the channel remain patent, but the cerebral hemisphere injected underwent atrophy. One such case has been subjected to microscopic examination and, aside from the possible existence of doubtful secondary degeneration, I am able to say that no trace of any lesion, hemorrhagic or inflammatory, was discoverable. The perivascular spaces were greatly dilated. The other, remarkable for the length of time the animal was kept alive—it was killed thirteen months after the operation—I have simply transsected the brain of at the line of injection. It exhibited at the time the fresh dissection was made the entire channel made by the needle, somewhat wider than the latter, but perfectly rectilinear, the shape of the point of the needle which

struck the capsule being readily recognizable. The walls of the channel appeared slightly buff-colored, and a rusty colored fluid filled it. Hardening has almost obliterated the outline of this channel, except at its entry point, but the atrophy of the caudate nucleus and thalamus and consequent collapse of the entire hemisphere is very evident. What is the cause of this atrophy? Is some such result to be apprehended from deep insertion of exploring needles in human brain surgery? I think not. The mass operated on is so much greater that the slight amount of displacement by the needle is of no moment. Still it were well to bear in mind that a species of irritative atrophy can be set up by such intervention, not due to gross lesions that could otherwise explain its occurrence.

In the course of a series of experiments, unfortunately interrupted, but which showed, as far as they went, that the more frequently the brain of the dog is interfered with by the insertion of indifferent and virulent substances in the brain tissue and subdural space, the greater its resisting power to injurious agencies which produced uniformly fatal results in dogs operated on for the first time. I found two cases of dwarfed growth and imbecility developed, which unfortunately terminated in death through neglect during my absence from the city. In one of these instances, a Newfoundland dog, the elongated cranial contour showed that the bilateral injection of zymotic material, although followed by no immediate results, had done symmetrically what the single injection on one side did in the dog whose brain is exhibited.

From my limited experience with human, and the observations cited from experimental pathology, I should be inclined to draw the following conclusions:

1st. That exploratory needles should never be introduced in the internal capsule, the contiguous ganglia, or the lateral ventricles, merely for exploratory purposes, unaided by positive clinical indications of the location of disease.

2d. That exposures of large surfaces of the brain are not

feasible in persons with feeble vascular walls, owing to the danger of intracerebral hemorrhage.

3d. That buttons of bone, reinserted under aseptic precautions, are, even in the event of non-union, entirely harmless.

4th. That in young persons, buttons of bone may become reunited to the calvarium even though perfect coaptation be not assured.

I should add that I have very recently experienced how desirable it is that, in the event of trephining for the relief of cortical irritation, on removal of the latter the button of bone be replaced.

A janitor, injured seven years previous by a fall of a beam a little to the left of the vertex, over the post-central gyrus and superior parietal lobule, had attacks of epileptiform derangement inaugurated by intense bursting pain at the spot indicated, and numbness followed by rigidity of the right leg. At my suggestion, Dr. Vanderpoel trephined at the spot in question. An adhesion to the dura requiring careful dissection was found, and for three months the patient showed a complete change of character, and remained free from seizures. At the end of that time, while arranging some bell-wires, one broke, and springing struck him precisely over the trephined spot with the point, inflicting a scalp wound.

That very moment the rigidity and numbness of the leg recurred, the headache returned, increased in intensity, and another epileptoid mental equivalent developed. Since that time there have been numerous recurrences, all marked by the same aura.

#### REMARKS ON DR. SPITZKA'S PAPER.

DR. GRAENE M. HAMMOND, of New York.—I agree with Dr. Spitzka that the button of bone removed in trephining should always be reinserted. I saw one of Dr. Weir's cases, in New York, in which he had removed from the right temporal region a piece of bone, operated on the cortex, and then replaced the piece of bone removed. Two months afterwards, when he exhibited the patient to the neurological society, the piece had become

solidly united to the cranium. I do not know whether that is the usual result or not, but in his case it was firm and made a good protection for the brain over the seat of the operation.

DR. JAMES HENDRIE LLOYD, of Philadelphia.—I would like to ask Dr. Spitzka if he has punctured the brain in infants, the subjects of hydrocephalus. I ask the question because recently a small infant was brought to me with the idea of having this done. I speak entirely apart from other considerations, as to whether or not such an operation would result in good, but simply with respect to injury to the brain, whether there is any special danger, how large a trocar should be used, what precautions should be taken, and also the location of the point where the puncture should be made.

DR. C. K. MILLS, of Philadelphia.—While I think it is undoubtedly a good thing to reinsert the button of bone removed in trephining in many cases, yet, from my experience in having the operation performed, as I do not perform the operation myself, it seems to me that there might be possible objections to it. In some cases, at least, as it seems to me, there would be a greater tendency to the occurrence of inflammatory conditions of the dura; that is, an increased liability to the development of a certain amount of inflammation from irritation. I think that it is well worth while to consider this point in connection with some of the cases. In cases where are found irregular conditions, so far as the bone is concerned, it might be sometimes objectionable to reinsert the piece removed. Dr. Spitzka does not mention the size of the piece removed in Dr. Hammond's case.

DR. HAMMOND.—It was about an inch square.

DR. MILLS.—I am unable now to give the exact size of the portion removed in one of my cases, but I will secure the exact measurements and forward them so that they can be placed on record. I am sure, however, that the portion removed was three or four inches in one direction and in the other direction perhaps three inches.

DR. SPITZKA.—For medical or surgical indications?

DR. MILLS.—Simply to meet surgical indications. The depression of bone was situated over the longitudinal sinus, and we believed that there were very strong indications for another operation, and the bone was removed by trephining twice and then using the rongeur. It is in conditions of that kind that it might be best not to attempt to replace the bone removed. In this case



it would have been desirable to restore the bone, but there was an irregular mass, and the question is whether there would not have been a recurrence of the conditions for which trephining was performed. One more point in Dr. Spitzka's paper interested me very much, and it was in connection with the third case. The third of his cases was highly successful so far as the surgical procedure was concerned. Two were successful so far as the results obtained, for which the operation was performed. In the third case, the patient is living and the mental condition is improved. But the fact, if I understood him right, that after one operation the animal stands the next operation better, brings him to the conclusion that the second operation can be performed with greater impunity than the first, and so on, the third and the fourth, that I should regard as an important practical matter as borne out by my observations in those cases referred to and in others not mentioned. In the first case the operation was the most serious which I have ever seen performed on the head, with the loss of large quantities of blood, and the patient was in the condition of violent acute mania, yet she made a perfect surgical recovery in a short time and is improved mentally. In two of the other cases, the operation was serious, and yet the patients had not an untoward symptom. The fourth patient died, but was almost moribund when the operation was performed.

DR. SPITZKA.—I should say, in justice to the surgeon who performed the operation and reinserted the piece of bone in the case described, that it was Dr. Charles McBurney, of New York. I am ignorant of the case cited by Dr. Hammond, but I think the real credit belongs to McEwen, who, while he did not reinsert a piece of bone, opened the way for it by sprinkling on bone dust or making bone grafts. My own attention was drawn to this forcibly by noticing that the bone dust from the trephine, when it dropped into the cavity, did no harm whatever. A large amount of bone dust in trephining may drop into the cavity and disappear.

With regard to the question asked by Dr. Lloyd concerning hydrocephalus, I should not hesitate in a case of internal hydrocephalus to insert a large canula under antiseptic precautions. I have not performed that operation myself, but I have seen it done successfully by Dr. Detmold, of New York. I have tapped twice, but they were cases of large hernias through defect in the skull, and the operation was performed without any difficulty whatever.

As is well known, we operate on spina bifida with antiseptic precautions and the use of Morton's fluid with perfect impunity.

With regard to dogs surviving the second operation better than the first, there may be a source of error in that statement, because the dog surviving the first operation may be a better animal than those in another series. But I got my impression from experimenting on 120 dogs. My first object in experimenting on these animals was not that of the paper, but for making observations in connection with experimentation on hydrophobia; to show to the profession and the public that there was a spurious creation of a hydrophobic scare. It was for this purpose that I repeated the experiments to see if I could obtain the results, and they were repeated and the result obtained again and again. The material which I used was of different kinds, and it is remarkable that in four cases I used such horrible substances that I could scarcely credit the claim I now make, as it seems to me, justifiably, had I before heard it from others. I used the filthiest of the street mud I could obtain, and the dog had never been operated on before, yet the wound did perfectly well. I should say that the button should be reintroduced only when the projection of the inner piece is removed. And I should have added the qualifying clause, when speaking of the largest removal ever made, "for medical indications." We are all aware that a large part of the cranium has been removed successfully in surgical cases.

DR. JAMES J. PUTNAM, of Boston, then read the notes of a rare case of

#### HYPEROSTOSIS OF THE CRANIUM.

E. L., 29 years old; married for ten years. Her father is living and well; her mother died during the patient's youth, and she can only remember that she had some trouble with her head for some years previous to her death. One sister, who died eventually with phthisis, also had a trouble with her head which patient believes to have been the same as her own. She died in Sweden while patient was in this country, and she cannot tell more accurately about her.

E. L. was in good health up to the time of her confinement with her first child, nine years ago. At that time, she began to have intense pain in the head, mainly at the

vertex, through the temples, and in the eyes. About two years later, she had a miscarriage; after that the pain became worse. The pain was at first intermittent, later rather continuous.

The broadening of the face and prominence of the eyes were first noticed about six years ago, and have steadily increased. There was no history of either rachitis or syphilis.

Her friends say she is still as intelligent as before, but this, of course, has not been fully tested.

Her teeth began to fall out three years ago, and are now all gone.

Two years ago, she began to have purulent discharge from both ears, and to lose her hearing. She is now perfectly deaf, and occasionally has discharge. She thinks she can hear her own voice, and that at one time this was not possible, but it is probable that in reality she only feels the vibration, for she also thinks she can hear footsteps.

Her gait has been tottering and stumbling for some time past, and she has even fallen to the ground, probably because she feels dizzy when walking. The use of the hands is good, though feeble, and she has never had pain in the limbs.

Her nutrition has maintained itself, and her appetite has remained good.

Previous to five years ago, her skin was white and fair; since then it has been growing dull and sallow, on the hands as well as the face. The catamenia have been absent for four years. When seen by me her physical condition was as follows:

Patient sits with mask-like face; lines of expression being almost obliterated. Lips scarcely move, if at all, in articulation; in fact, lower lip hangs down perfectly flaccid, unless when raised by hand, when it retains its place purely by adhesion.

The eyes so prominent that at least half the globe seems to project beyond lower lid. They are continually open

from paralysis of the orbicular muscles. Motion of both globes perfect and harmonious.

There seems to be complete paralysis of upper as well as lower branches of facial nerve. Face and head have broadened in a remarkable manner; the skull being especially broad about level of eyes, between eyes and ears.

Circumference of head, 59 cm.; greatest breadth, 16 cm.

Color of skin of face and hands sallow.

When asked to protrude tongue, first puts it out towards right, and to less extent than normal; but when urged to put it towards left does so with apparent ease, but at best does not get it beyond her teeth.

Speech very imperfect, scarcely intelligible, from failure, apparently, to use lip and tongue.

She can use both arms and legs, but rather feebly.

Husband thinks that she uses one eye better than other, but ophthalmoscope shows no neuritis, congestion, or atrophy, and vision is apparently perfect.

Cornea dry, on account, evidently, of the lids not closing. Pupils small.

Purulent discharge from both ears. Nose looks as if jammed inwards, evidently on account of the prominence of forehead and cheeks. Bones of antrum not very prominent.

Electrical reaction. Faradic reaction absent in both facial nerves, and degeneration reaction present in muscles of lips, forehead, and cheeks. Only a weak current could be used on account of sensitive condition of patient.

DR. PUTNAM also presented a

#### SARCOMATOUS TUMOR OF THE BRAIN

with the following history:

Mrs. F., married, in good circumstances, 38 years old; in good nutrition and in apparently good health when first seen (February 9th, 1886).

No family history bearing on present illness; tendency

to periodical headaches lasting through one day and passing off at night-fall, which patient thinks she inherited from her father. About eighteen months before my examination, these headaches increased markedly in frequency, but did not change in character.

The whole duration of illness, *i. e.*, from the time when the first symptoms—possibly attributable to her disease—were noticed until death, was about twenty-one months; but no symptoms unequivocally due to the disease were noticed until about four and a half months before her death.

The first of the possible symptoms was the increase in the frequency of the periodical headaches.

The first unequivocal symptom was a terrific headache, which lasted two days, and ushered in an almost unbroken series of headaches which continued until death.

The severe attack began just before a catamenial period, and the flow ceased after two days instead of lasting five, as was customary. The headaches were mainly frontal, but the pain sometimes spread all over the head and into the nuchal region. She rarely suffered much during the night, but greatly on waking in the morning; and she had had a few severe attacks of vomiting in the morning.

Besides the headaches, she began to have, at this time, slight and vague sensations of vertigo, especially on stooping, often accompanied by a feeling as if the floor were rising to meet her.

The gait was not staggering, but there was a slight loss of balance on turning suddenly.

Vision was slightly indistinct (o. d. .06; o. s. 0.3, H. D). and well-marked optic neuritis was present.

There were no localizing symptoms of cerebral disease.

The mental condition was good, except that she showed a sort of indifference and light-heartedness with regard to her symptoms and her condition, which is, perhaps, attributable to her disease. Except for these symptoms, the patient appeared at that time to be in excellent health,

and it is interesting to note, in view of the seat of the tumor, and the fact that it probably began to increase pretty rapidly in size two months before my examination, that my notes speak of her as appearing rather unusually talkative at her first visit.

From this time the patient became gradually worse until her death, which occurred two and a half months later. The manner in which the increased pressure showed itself was rather interesting. For six weeks before death she lay nearly unconscious, and apparently without much sign of pain, unless it was on this account that she kept her right hand constantly at her head and yet she could be roused to make more or less response up to the last day before her death.

Even before becoming so stupid, her power of conversation had been getting more and more limited, and this was somewhat out of proportion to the mental limitation which was likewise progressing. A single word or phrase of exclamation or assent, such as "I declare," or "don't say," would be made to serve for answer to every sort of question, and, as a rule, she ventured no remarks on her own account. At the same time such expressions as she did use were used correctly, and in her delirium she talked coherently about her home affairs. It is interesting to note that such localizing signs as there were, were referable to both sides of the brain. Thus, the tumor being on the left side, she complained a great deal of pain in the two middle fingers of the left hand, running upward to the elbow, and at one time she used the left hand less than the right.

There was a paresis of the buccal muscles of the right side of the face, possibly the upper also to some extent, with slight deviation of the tongue to the right.

The pulse varied in frequency between the outside limit of fifty and seventy-five, but became weaker. The respiration did not change much in frequency or rhythm, except that during one night, by the description of her friends, it was of Cheyne-Stokes' character. It was, however, labored and heavy much of the time during the last weeks of life.

The temperature remained normal so long as I saw the patient, which was within a few days of her death.

The examination of the head was made thirty hours after death. I will speak only of what was abnormal.

The convolutions were flattened so that all trace of rounding was obliterated, and the sulci appeared as lines drawn on a flat surface. The tumor was first seen, after laying back the flaps of the dura, as a dark glistening mass which proved to be a cyst of the outer and upper portion of the tumor, filled with blood and detritus, and occupying the position of the caudal extremity of the second and third left frontal convolutions. The remainder of the tumor was much firmer and lay mainly beneath the frontal lobe, reaching toward the median line so far as to push the left olfactory nerve somewhat to one side. The only attachment of the tumor was to the dura mater covering the left wing of the sphenoid over a space somewhat larger than a nickel cent. The attachments were broken without difficulty, leaving a roughened surface, beneath which the bone was smooth. The mass had forced the lips of the Sylvian fissure widely apart and the frontal lobe was reduced to about two-thirds its natural size. The third frontal in particular was thinned, and softened by the pressure and œdema. There was no localized or inflammatory softening. The cerebral arteries were pervious and supplied the tumor in part with blood; but except for this there was no connection between the tumor and the brain.

The surface of the brain was dry and blanched.

As soon as the tentorium cerebelli was cut, a large quantity of clear fluid gushed out and the ventricles also contained fluid, but were not distended.

The tumor, after hardening in alcohol, displaced 100 ccm. of water.

It proved on microscopic examination to be a sarcoma, and in spite of its size it seems not impossible that it could have been removed if its position could have been ascertained.

It is, of course, just in the case of these slowly-growing

benign tumors that localizing signs, which might serve as a guide for operation, are least often found. The question may, however, be raised whether, when, as here, unmistakable signs of a slowly progressive growth are present, and no sign of constitutional taint can be found, the presence of such a sign as facial paresis, which was detected in this case several weeks before death, ought not sometimes to be considered as an indication for an exploratory operation, in the hope that just the state of things might be present which has been described. Thermometric observations were not made. I will only add that this case is another instance in proof, if further proof be needed, of the fact that the absence of susceptibility to iod. potassium is not a necessary indication of syphilis.

The dose was carried, without any material difficulty, to two grammes, and could have been pushed further had the course of the symptoms offered any encouragement for so doing.

#### REMARKS ON DR. PUTNAM'S PAPER.

DR. SPITZKA, of New York.—There is one source of error in the explanation of the symptom of pain in the two fingers mentioned by Dr. Putnam. I saw a remarkable illustration recently in one of the recorded cases in which there was an easily localized surface tumor of the brain with complete paralysis of the opposite arm, preceded by the same sarcomatous growth of the brachial plexus as existed in the brain, and it is possible that the symptom of pain in the two fingers in Dr. Putnam's case was not due to the cerebral tumor. There is this difference between the symptoms of the cortical and spinal lesion. In the former we have numbness along the course of the whole hand and forearm and rarely in the distribution of one nerve, and so long as the peripheral nerves were not examined we are not certain how the symptom is to be interpreted. If we have facial paralysis and ulnar-nerve disease at the same time, we should expect that they were due to the same lesion.

I would ask Dr. Putnam if he had encountered in literature a remarkable case of hyperostosis in which the disease was so extensive that the orbital cavities were obliterated, the antrum of Highmore almost completely closed, and the skull porous. The



association of double otorrhœa is remarkable and interesting in his case.

DR. PUTNAM.—I recognize perfectly well that in the absence of a post-mortem examination of the entire body, the possible, perhaps probable, justice of Dr. Spitzka's criticism. I have seen a patient, however, who had a tumor of the brain, lasting many years, and eventually disorganizing both sides of the brain, who presented for a long time absolutely no symptoms except pain and numbness of one wrist, not following the course of any particular nerve. In the case reported, the pain was in the two middle fingers, and unless I am mistaken, peripheral pains, not occurring in the course of single nerves, and yet not segmented, are among the symptoms of cerebral disease.

(To be continued.)

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## Editorial Notes and Miscellany.

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At the last meeting of the German Medical Congress, Professors Nothnagel and Naunyn made a report upon the present state of the question of cortical localization which deserves more than a passing notice. The report is in many respects an interesting one, and an additional one need not be looked for for many years to come.

The most important conclusion at which Nothnagel arrives is, that, as far as *human pathology* is concerned, cortical localization is a settled fact. He refrains from expressing any opinion regarding the conflicting views of physiologists. But Nothnagel is not willing to accept the ordinary views regarding psycho-motor centres, for he shows that a patient can be possessed of the intention of performing a movement without actually being able to perform it. In other words, that the memories of previous movements are not necessarily stored in the same cells from which definite movements are started. Applying the same argument to the visual centres, Nothnagel claims that a person may become totally blind from the destruction of both so-called visual centres, but that he may still retain the memories of former visual impressions. Such terms as psycho-motor centres should therefore be dropped. The most that can therefore be said of a centre—of a motor centre, to

instance—is that it is a special area which must be uninjured, if certain movements are to be performed properly ; but that the motor impulse itself may originate elsewhere in the cortex.

Nothnagel gives a very complete summary of the various forms of visual disturbance that may be due to cortical lesions : he agrees with Seguin in relegating the actual visual centre to the *cuneus*, but he would wish to include with it the first occipital convolution. In the remaining portions of the occipital lobe, the mass of visual memories is housed ; as for the motor centres, these are situated in the central convolutions and the paracentral lobule, but possibly the “frontal” portions of the parietal convolutions may have something to do with motion. There is some reason to think that the muscular sense is dependent upon the parietal lobes. Nothnagel is not willing to risk any positive statements regarding sensory localization in the cortex. Many other questions are discussed ; the paper is undoubtedly the most masterly summary that has yet been presented on this vexed subject.

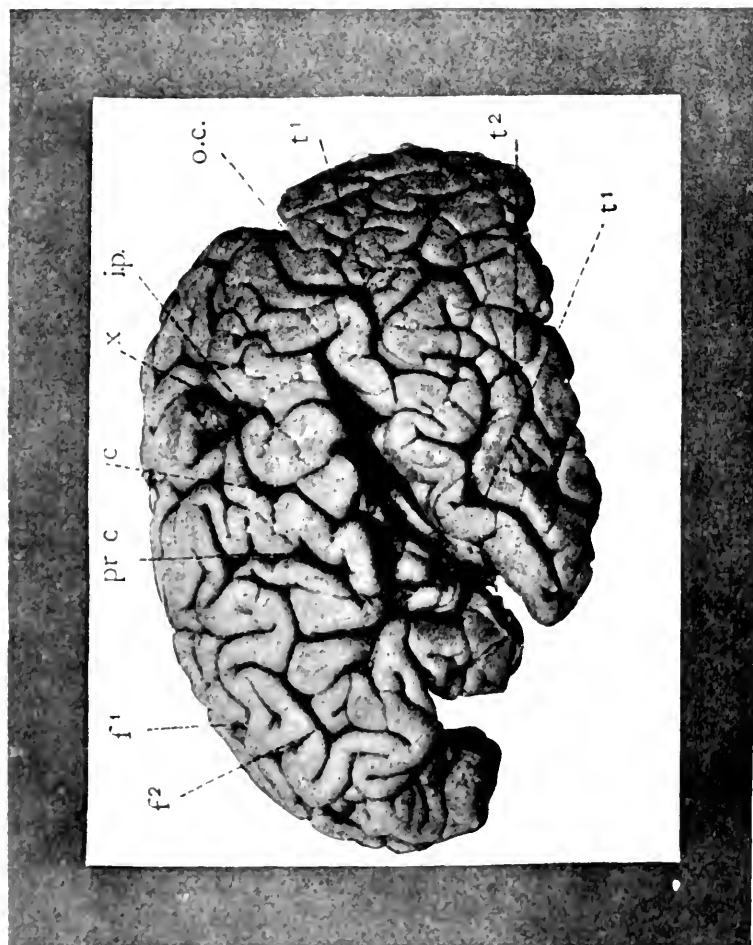
Prof. Naunyn treats of the subject of cortical localization with special reference to aphasia ; he has examined the literature very carefully (he has not apparently had access to American literature), and has noted the lesions found in these cases upon a chart of the brain.

As a result of this study, he finds that there are three distinct sets of lesions in the various forms of aphasia.

The first set is confined chiefly to Broca's convolution (pure motor aphasia) ; the second set to the posterior two-thirds of the first temporal convolution (aphasia with word-deafness) ; the third set to the transition from the angular gyrus to the occipital lobe (aphasia with word-blindness). This proves conclusively that, as the present writer has long since been in the habit of teaching, instead of there being any *one* speech centre, widely different parts of the cortex subserve the functions of language. The amount of information which Prof. Naunyn has been able to condense into the short resumé is quite astounding.



PLATE I.



THE  
Journal  
OF  
Nervous and Mental Disease.

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Original Articles.

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ON ARRESTED CEREBRAL DEVELOPMENT,  
WITH SPECIAL REFERENCE TO ITS COR-  
TICAL PATHOLOGY.<sup>1</sup>

BY B. SACHS, M.D.,

NEW YORK.

OUR knowledge of the pathological substratum of the various forms of mental derangement is still very imperfect. In the majority of cases, there may be no marked changes in the structure of the brain; or, if there be any changes at all, they are entirely beyond our ken, and cannot be made out by our present methods of investigation. As mental pathology is in its infancy, it is but natural that we should first seek for structural changes in those conditions in which the departure from the normal is greatest, in which the mind is disturbed, as a whole, and not merely with reference to a single part or faculty; though I shall at once declare my belief that derangement of a part of the mind means disorganization, more or less complete, of the entire mental mechanism.

While we are even now in possession of many facts concerning the morbid structural changes in dementia paralytica, changes that accompany the complete *dissolution* of a fully developed, and once normal mind, we have busied ourselves but little with the morbid changes that often

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<sup>1</sup> Read before the American Neurol. Assoc., July, 1887.

affect the brain, and consequently the mind also, when both are yet undergoing the process of *evolution*. These cases of retarded development, of idiocy, of mental imbecility, call them what you will, seem to me to possess a deep pathological and physiological interest. From the pathological changes found in these cases of extreme mental defects, we are entitled to draw an inference regarding the normal function of those nervous elements here found deficient, and we may well argue with regard to such broad facts as an absolute lack of mentality, although it may be a long time to come before we shall be able to explain the morbid mechanism underlying fixed delusions, hallucinations, and the like, or to state exactly what the structural changes are in paranoia, in circular insanity, and in other grave mental troubles.

The condition which I have the privilege of discussing before you to-day represents not only such changes as come about in the process of evolution, but represents changes of the earliest period of infantile development.

Much has been written upon idiocy and allied conditions from the clinical point of view, but pathological and pathological-anatomical observations are surprisingly few and far between. And those who give gross morbid changes fail to refer to the histological changes either in the cortex or other parts. Thus Bourneville,<sup>1</sup> who has made an excellent contribution to the study of this subject, refers in only one of five cases to changes in the cortex. Brückner<sup>2</sup> has given the most detailed account of the histological changes in the cortex with which I am acquainted. His was a case of what is known as tuberous sclerosis of the cortex, and concerned a patient 22 years of age. The pathological changes underlying these conditions of idiocy are undoubtedly as varied as the clinical manifestations themselves; for the present we designate these affections by broad clinical terms; later on we may

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<sup>1</sup> Bourneville ("Sclérose tubéreuse des circonvolutions cérébrales"). Arch. de Neurologie, vol. i.

<sup>2</sup> Brückner, "Ueber multiple tubéreuse Sklerose der Hirnrinde" (Arch. f. Psych., vol. xii.).

be able to differentiate between them, and to give to each condition its proper pathological designation. Looked at in this way, the title of my paper is altogether too comprehensive. The changes which I have to report upon to-day are a few of the many changes which may give rise to similar clinical mental phenomena.

Before presenting the history of the case, I must acknowledge my indebtedness to Dr. I. Adler, of New York, through whose kindness I was enabled to observe the case closely, and with whom I shared the responsibilities of treatment; and to my friend, Dr. van Gieson, who was kind enough to supply me with normal material of the same age for comparison, and who, during an unexpected absence from the city, assisted me in the work of cutting and staining.

The following is the history of the case: The little girl S., who was but two years old at time of death, was the first-born of young and healthy parents. In the families of both parents insanity is not unknown; on the mother's side there is a strong hereditary predisposition to mental disease, and several near relatives of the father have developed various forms of insanity within recent years. During the fifth month of pregnancy, the mother was thrown out of her carriage, but did not sustain any serious injuries; the child was born at full term, and appeared to be a healthy child in every respect; its body and head were well proportioned, its features beautifully regular. Nothing abnormal was noticed until the age of two to three months, when the parents observed that the child was much more listless than children of that age are apt to be; that it took no notice of anything, and that its eyes rolled about curiously (there was evident nystagmus). Allowing for some very slight vacillations, the child remained in practically the same condition up to time of death. The condition was characterized as follows: The child would ordinarily lie upon its back, and was never able to change its position; muscles of head, neck, and back so weak that it was not able either to hold its head straight or to sit upright. It never attempted any volun-

tary movements; movements that were made were in obedience to peripheral stimulation. All the muscles were extremely flaccid; all reacted perfectly to both forms of current. The child would close its hand upon the finger of the examining person, but objects placed in its hands were quickly dropped. The child as it grew older gave no signs of increasing mental vigor. It could not be made to play with any toy, did not recognize people's voices, and showed no preference for any person around it. During the first year of its life, the child was attracted by the light, and would move its eyes, following objects drawn across its field of vision; but later on absolute blindness set in.

Dr. Knapp, who made several ophthalmoscopic examinations of this case, reported the following unusual condition, at the seventeenth meeting of the Heidelberg Ophthalmological Society. The report may be found in the Proceedings of this meeting. Dr. Knapp there says: "Child two to three months; nystagmus vibratorius; pupils contracted as is usual with children at this age. Media clear, optic nerve discs pale. Fovea centralis, of a cherry red color, was surrounded by an intense grayish-white opacity. This opacity was most distinct in the vicinity of the fovea centralis, and for some little distance around it, but faded away gradually into normal retinal field."—Dr. Knapp at first gave a favorable prognosis, except as regards central vision, more particularly as there appeared to be for some time a slight improvement in vision. He could not then, and is not now ready, to give an explanation of this condition.

But two cases of this sort of retinal changes had thus far been reported, by Magnus and Goldzieher, and neither of these authors has any explanation to offer. Dr. Knapp, in private conversation, hinted at a developmental defect. Unfortunately, the eyes could not be removed after death. Dr. Knapp empowers me to add that "a further examination in May and June, 1886, revealed great changes. Child totally blind, optic nerves completely atrophied (discs as



white as paper, with scarcely a trace of blood-vessels). Macula lutea essentially as before."

By way of anticipation, it may be remarked that numerous longitudinal and vertical sections of both optic nerves were variously stained and examined, but that no morbid changes could be made out. Blindness must, therefore, have been due either to the retinal changes, or to the deficient cortical condition, or to both.

Hearing seemed to be very acute; there was unusual hyperexcitability to auditory and tactile impressions; the slightest touch and every sound were apt to startle the child. The child never had convulsions, not even while teething; no marked rigidities at any time. The child never learned to utter a single sound; if left to itself it would occasionally make a low gurgling noise. Bodily functions normal, excepting the frequent recurrence of bronchial troubles and feebleness of its digestive powers. At the age of one it had a severe attack of diphtheria from which it rallied in the course of a few weeks. The child developed unusually high fever with every disturbance, however slight, of its bodily functions. In the way of treatment nothing was recommended but careful nursing and feeding, tonic treatment with malt and the like; phosphorus was given in small doses for a time, and the peripheral muscles and nerves were alternately galvanized and faradized, more in the hope of exciting cerebral activity in a reflex way than of benefiting the nutrition of the flaccid parts.

There were no distinct evidences of inherited or acquired syphilis and none of rachitis.

During last summer (1886), the child grew steadily weaker, it ceased to take its food properly, its bronchial troubles increased, and finally, pneumonia setting in, it died August, 1886.

Immediately after its death, the child was brought to the city, and yet twenty-nine hours had elapsed before the autopsy could be made.

*Autopsy.*—The autopsy was confined to an examination of skull, brain, and abdominal viscera. The body was in a

state of extreme emaciation ; all muscles relaxed. The skull was thick, and skull cap unusually heavy. Outer and inner surfaces smooth and showed no unusual appearances or impressions. Skull symmetrical ; left frontal fossa a trifle deeper than right ; large fontanelle very nearly ossified. A large organized clot was found in the superior longitudinal sinus ; there was some thickening of the dura to either side of the sinus, some slight adhesion over the upper portion of the precentral and over the left temporal convolution, but even here and over the entire surface of the brain the pia could be easily removed without injuring the parts below. There was an œdema of the entire convexity ; unusual pallor of the convolutions ; no marked increase of the fluid in the lateral ventricles. Freed of its dura, the brain weighed exactly two pounds (one thousand grams). Blood-vessels appeared normal and had normal distribution. I may state at once that the cortex was hard to the touch, and that the knife grated in removing a small portion of the cortex for immediate examination. This grating was due to small calcified plates. On superficial inspection, the great breadth of the fissures, the corresponding narrowness of the convolutions, and the unusual exposure of the left island of Reil were very apparent. A detailed examination of the larger ganglia, of the pons, medullary, etc., will be made and will be reported upon later on.

The spleen was enlarged and the liver hard, but no evidences of hereditary syphilis.

#### EXAMINATION OF BRAIN.

The brain was immersed at once into Müller's fluid, and as soon as hardened the brain surfaces were photographed.<sup>1</sup> By comparison with the paper<sup>2</sup> which our retiring president read last year, you will recognize certain departures

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<sup>1</sup> I am indebted to Mr. O. G. Mason for the original photographs, but one of which is reproduced in this article ; all four photographs were exhibited at the meeting of the Association.

<sup>2</sup> C. K. Mills, Presidential Address (*JOURNAL OF NERVOUS AND MENTAL DISEASE*, vol. xiii., 1886).

from normal fissuration which are indicative of inferior brain development.

*Examination of Brain Surfaces.*

Left hemisphere, outer surface. (Plate I.)

The most striking features are the great depth of all fissures, and the comparative simplicity of fissuration, particularly in the frontal lobes; the great exposure of the island of Reil due to the retraction and narrowness of the surrounding convolutions. The central fissure (*c*) is bifurcated and is clearly confluent with the Sylvian fissure which is broad and long. The first temporal fissure (*t. 1*)—supertemporal, Wilder—would be continuous high up into the parietal region, but for a slight bridging convolution. The parieto-occipital fissure is unusually distinct and in the occipital lobe the three fissures are easily traced. In the frontal lobe, the first and second frontal fissures are well marked, while the second forms the long branch of a zygial formation according to Wilder. The convolutions appear alternately narrowed and broadened; this is particularly true of the first temporal and precentral convolutions. The gyrus angularis is scantily developed.

The mesial surface of left hemisphere exhibits the confluence of the parieto-occipital, the calcarine and hippocampal fissures. The collateral fissure of Wilder well marked. The callosomarginal fissure well defined though shallow. The præcuneus massive, the cuneus of normal size.

Right hemisphere—*outer surface.*

Here the conditions approach much more nearly to the normal. The island of Reil is scarcely exposed; the fissure of Sylvius of normal breadth and length; the central fissure is confluent with the fissure of Sylvius. The first temporal convolution is *continuous* into the parietal region, and there is a distinct though very narrow angular gyrus. Wilder's interparietal fissure is distinct; in both the occi-

pital and frontal lobes, three typical fissures can be made out; there is an undoubted medifrontal (Wilder) fissure which could not be traced on left side. The parieto-occipital does not form as distinct an indentation as on left outer surface.

Median surface.—The parieto-occipital, calcarine, and hippocampal fissures are confluent; the collateral fissures well defined; the entire mesial surface is divided into small blocks by numerous secondary fissures. Cuneus and præcuneus of normal development.

#### *Microscopical Examination of the Cortex.*

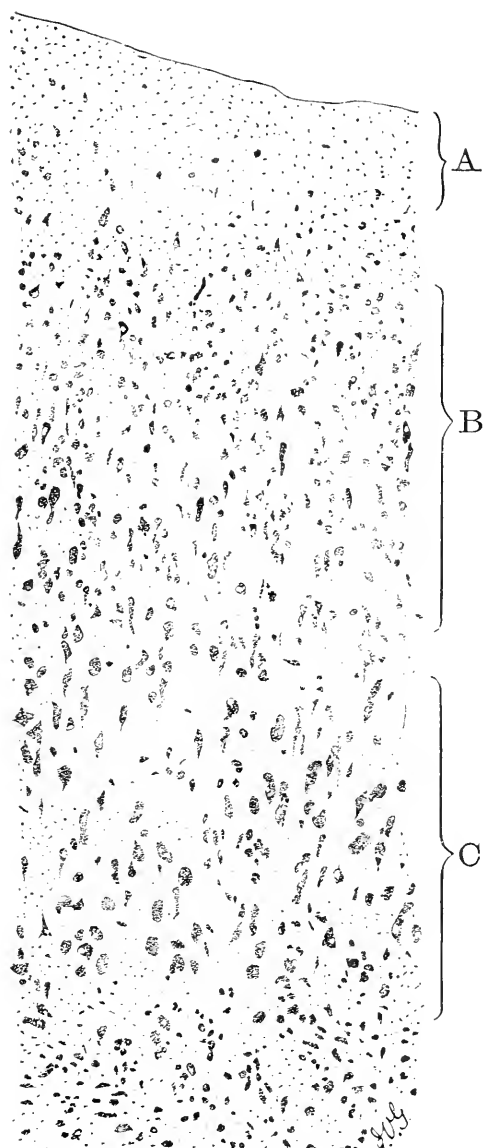
The brain surfaces, after they had been thoroughly hardened in Müller's fluid, were cut up into small blocks for histological examination. Sections from the frontal lobes, the motor zones, the base of the third frontal convolution, from the first temporal convolution, and from the occipital apex of both hemispheres have been examined. The cuneus was unfortunately too brittle to permit of section cutting. From the portions thus far examined, it is fair to infer that the changes to be described affect equally every part of the brain surface. The plates herein given represent the changes as seen in sections from the first temporal convolution of the left side. These specimens were stained according to the acid fuchsin method, others were stained with Weigert's two hæmatoxylin methods, and with ammoniacal carmine. You will note that the cellular elements exhibit the same changes, whatever staining method we employed. On the drawing, most carefully made by Dr. Van Gieson, and in these specimens<sup>1</sup> the following conditions may be noted.

We are able to distinguish the external barren layer, the layer of small pyramidal cells, the layer of the large pyramids, and perhaps a trace of Meynert's fourth granular layer. Examining these sections, very marked

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<sup>1</sup> Demonstrated at the meeting.

PLATE II.



changes will be observed in the structure of the small and large pyramid cells. In my search through the entire brain I have not come across more than half a dozen, if as many, pyramid cells of anything like normal appearance.

## PLATE III.



The fewest large and small pyramid cells show well-defined processes. The contours are rounded, and the cell substance exhibits every possible change of its protoplasmic substance. In some there are a distinct nucleus and nucleolus, surrounded by a detritus-like mass; in many

the nucleus and nucleolus are entirely wanting. All these varied changes can be studied best with the acid fuchsin method; in Weigert preparations, the whole pathological cell mass takes up the stain deeply, and it is not always easy to distinguish the nucleus and cell-body. Glancing through the sections, you will also observe that a few of the cells turn their apices downward instead of upward, thus exhibiting a change to which Brückner refers as occurring in his case of tuberous sclerosis and to which no pathological significance is to be attached.

Plate III. exhibits these changes under a very much higher power. In some cells a partly normal and a partly pathological character of the cell-body is visible. In the neuroglia, I have not been able to prove any changes; there is certainly no sclerosis visible in any part I have examined. The white fibres have not undergone morbid changes, but on Weigert specimens they cannot be traced as far towards the periphery as in the normal cortex; the transverse fibres in the outer barren layer could not be made out. There is no evidence whatever of any previous encephalitic process. No infiltration around the blood-vessels; in fact no changes in any of the blood-vessels of the cortex. At the meeting, doubts were expressed whether there was not a paucity of blood-vessels. I have paid special attention to this point, and am now convinced, after examining a very large number of sections from every part of the cortex, that these capillary vessels are of normal calibre and as numerous as in corresponding sections of the normal brain. Nor is there any proliferation of the nuclei of these cells in the walls of the blood-vessels. We have then a simple change affecting the cells and possibly the white fibres only, and the question remains to be decided whether there is mere arrest of development, or an arrest of development the result of a previous inflammatory process. There is nothing in support of the latter proposition, and everything in favor of the former.

I cannot find any evidence of distinct degenerative changes in the cells, and it would seem to me that, if the

process were one that had set in after the cells had already matured, we should find some, and many more cells than we actually do, exhibiting a more complete formation than any to be found on the specimens before you. You will note also that there were no gross changes such as are frequently held responsible for insufficient development: there is no evidence of hydrocephalus internus, of a general or a multiple tuberous sclerosis; no traces of a preceding encephalitis.

We have here an agenetic condition pure and simple, affecting the highest nerve elements. As to the cause of this agenetic condition, I am not willing to speculate. I repeat that syphilis is excluded, at least not proved, that there is strong hereditary predisposition to mental troubles, and that there is the etiological factor of traumatism in the case. As the foetal circulation is easily affected by the slightest disturbances, and the proper nutrition of the most highly differentiated organ of the body may in this way have become impaired, we cannot afford to overlook the factor of traumatism.

#### EXPLANATION TO PLATES I., II., AND III.

##### PLATE I.

Outer aspect of surface of left hemisphere showing the exposure of the island of Reil, and great breadth of fissure of Sylvius.

X denotes region from which first block of cortical tissue had been removed for histological examination.

C, central fissure or fissure of Rolando.

*prc*, precentral fissure.

*i. p.*, interparietal fissure.

*oc*, occipital; parieto-occipital fissure.

*t*<sup>1</sup>, *t*<sup>2</sup>, first and second temporal fissures.

*f*<sup>1</sup>, *f*<sup>2</sup>, first and second frontal fissures.

Other explanations in text.

##### PLATE II.

× 70 diameters.

Section from first temporal convolution; specimen stained with acid fuchsin; drawing made with especial reference to changes in the cells.

Divisions A, B, C, correspond about to layers of superficial neuroglia, of small pyramid cells and of large pyramid cells. Below C is fourth granular layer (Meynert).

It will be noted that with this low magnifying power, the changed appear-



ance of the pyramid cells can be made out. The contours of the cells are altered, the pyramidal shape is often widely departed from; the cell body is altered, and occasionally shows distinct lacunæ.

## PLATE III.

× 500 diameters.

Section of first temporal convolution, representing a portion of division C under much higher magnifying power. Pyramidal cells have lost their normal shape. The cell body has a homogeneous but altered appearance; nuclei either absent or distorted; smaller cells and neuroglia cells with distinct nuclei; section of capillary vessels normal, in the upper right hand corner a distorted cell mass with pericellular space around.

## NERVOUS AND MENTAL DISEASES AS INFLUENCED BY THE CLIMATE OF COLORADO.<sup>1</sup>

By J. T. ESKRIDGE, M.D.

ABOUT two years ago, I determined to study the effects of Colorado climate on nervous diseases. I tried to begin my observations and inquiries without having formed any preconceived opinions of the subject. I soon found that many physicians of undoubted skill, practising medicine in the State for a number of years, were undecided as to the rôle played by Colorado climate *per se*. All the physicians there with whom I talked seemed to be agreed that the climate has a modifying influence, but to what extent, and in what particular respect, opinions were not so unanimous. Several excellent physicians, who have resided in Colorado a number of years, have refused to express an opinion, because they are still unable to formulate any in regard to the subject. It is just to say that most of those who hesitated to express an opinion have pleaded the excuse that they have paid no special attention to diseases of the nervous system while practising in the eastern States, and were consequently unable to draw just comparisons from their observations in mountainous regions. On the other hand, I must add that not all who have responded to my questions claim any special attainments in the study of nervous diseases; but they are educated men, on whose observations and honesty I can rely. I have been fortunate enough to find in Colorado a number of physicians who

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<sup>1</sup> Read before the Philadelphia Neurological Society.

appear to have formed decided views regarding the influence of the climate on nervous diseases.

In studying the modifying influence of Colorado climate, it is important we should bear in mind that other agents than climatal may have something to do with difference in the manifestation of disease in different places. To determine the sole influence of climate, we must be careful to exclude the influence of the difference in the habits of the citizens. Among the inhabitants of Colorado, we find more leisure in many places there, and a greater tendency to keep late hours and indulge in various dissipations than is common further east. Many go to Colorado in search of health, and the separation from relatives and friends, added to the enforced idleness, is a source of worry and a certain amount of nervousness. Some go to better their fortunes, and for these, investments in mines and various other uncertain speculations cause anxiety and unwonted excitement. Many who had lived quiet lives and kept regular hours for rest and eating in the eastern States, go to Colorado, over-indulge in the use of alcohol and tobacco, and try their nervous systems by late and irregular hours. After allowing for all the modifying influences, exclusive of climate, I feel confident that a careful comparison of certain nervous disturbances at sea-level with those met with in high and dry mountainous regions, a difference will be found to exist; but the difference is much less than the exaggerated statements made by the laity there, concerning the influence of Colorado climate on the nervous system, would at first lead us to believe. That among the people of Colorado we have more of what is termed nervousness than exists in the same number of inhabitants at sea-level there can be no doubt; but consumptive invalids form a greater proportion of the population of the former than of the latter. It is because of the great difficulty in judging of the influence of the climate of Colorado on nervous diseases, both organic and functional, that so many experienced physicians there have hesitated to give an opinion. During the last eighteen months, I wrote to a number of phy-

sicians at various places in the State requesting the result of their experiences with nervous troubles there, in comparison with the same at low altitudes. Places from which I have obtained replies vary in elevation from four thousand feet to ten thousand. I visited the State Insane Asylum at Pueblo, and through the kindness of the superintendent, Dr. Thombs, I was permitted to study the cases under his care. During a residence of three years in Colorado, I have seen a number of cases of nervous troubles. Most of the time I have remained at Colorado Springs, which has an altitude of six thousand feet, but I spent eight weeks in Manitou Park, at an elevation of between seven and eight thousand feet, and a short time at Greely, whose altitude is about forty-five hundred feet.

Under the various nervous troubles of which I have been able to obtain the opinions of physicians in Colorado, I will give their replies to my queries, together with some comments of the writer. Some of the statements made in this paper may not stand in the light of further observation, and it is hoped that this effort to study the effects of Colorado climate on the diseases of the nervous system may stimulate others to pursue the subject further.

Insomnia.—In speaking of insomnia, Dr. W. H. McDonald, of Pueblo, but formerly of New York, where he practised eight years, paying special attention to diseases of the nervous system, says: "Insomnia is less frequent and a far less serious trouble here than in the east. The early effect of this climate upon one coming to it is decidedly soporific. Sleep is notably profound and prolonged."

Dr. B. P. Anderson, of Colorado Springs, states: "In most cases of insomnia coming under my observation, this climate has exerted a salutary influence. Few cases have failed to find relief, and these few exceptional cases not deriving benefit from the climate have been due to organic lesions which subsequently terminated in grave brain affections. In all cases dependent upon over-work, hyperæmia, or reflex troubles, the climate has, in my experience, proved to be of the utmost benefit."

Dr. W. M. Strickler, of Colorado Springs, writes : "I have found that good and refreshing sleep is easily obtained. In only one instance in an experience of seventeen years in Colorado, have I advised a change to a lower altitude on account of insomnia."

Dr. M. H. Sears, of Leadville (altitude about 10,000 ft.), states : "I am firmly of the opinion that the high altitude of Leadville, where my observations have been made, has a marked effect on diseases of the nervous system. Cases of insomnia coming here from eastern localities, I believe, after a few weeks, show decided improvement without the use of hypnotics. I have observed in a number of instances, complaint, on the part of patients, of inability to obtain sufficient sleep. This in time wears off to some extent, varying of course with different individuals, as they become acclimated. My remarks apply more particularly to those persons in whom there is a determination of blood to the head from mental over-application, worry, etc., but in whose nervous tissues there is no degeneration — the blood over-supply to the brain being the sole cause of the insomnia. I believe the improvement in these cases is due to the lessened atmospheric pressure, and consequently increased amount of blood in the superficial capillaries. The pressure here is about eleven pounds. I have had some experience with chronic alcoholics, and if anything, their insomnia is increased. I recall several who could not obtain more than three or four hours' sleep out of the twenty-four, for days at a time. As regards insomnia in chronic, but slight, inflammation, congestions, etc., I have not noticed any particular effect of the altitude, possibly because medication has cloaked its influence, but the same causes, acting in simple insomnia as above suggested, must act in these conditions also. With insomnia from other causes, viz., cerebral tumors, etc., I have had no experience, nor have I heard of a case of tumor of the brain occurring here."

The writer has talked with several physicians of Colorado Springs, and of other places in Colorado, and the almost universal testimony has been that, for the majority

of persons, especially for consumptive invalids, sleep is more easily obtained, more continuous, and more refreshing in Colorado than in the eastern States. The tired, illy nourished and over-worked person who had spent sleepless nights east, goes to Colorado and finds, as his nutrition improves, that sleep is prolonged and unusually refreshing. Cool nights, throughout the summer season as a rule, enable persons to get much more sleep and rest in Colorado than can be obtained at sea-level during this portion of the year. Some on going to Colorado are unable to sleep well for a few nights, or perhaps weeks, whilst others get prolonged and refreshing sleep from the first. Those belonging to the latter group are much the more numerous. Those whose sleep is disturbed on first going to places of considerable altitude, usually enjoy a sufficient amount of sleep for several months after they begin to rest well, but I doubt whether these are ever able to sleep as much as those who rest well on first going to high mountainous regions. There is a popular, and almost universal belief among the laity, and physicians share this opinion, that one "wears out the good effects of the climate after a few years' continuous residence in Colorado." I am firmly convinced, both from observations and from inquiries among those who have resided there a considerable length of time, that there is a great deal of truth in this prevailing opinion. Those who lead idle or sedentary lives are, I think, more liable to become sleepless after a considerable stay there than those who keep profitably employed in work that requires more or less exercise. Much severe mental work at high altitudes would be, I think, more likely to be followed by sleeplessness than the same done at sea-level. Tobacco, alcohol, tea, and coffee, if indulged in immoderately, apparently injuriously affect sleep more at high altitudes than the same indiscretions do at low elevations. Whilst the majority of persons who go to Colorado get refreshing sleep for a number of months, and in some instances for years, yet there are a few nervous, hysterical individuals who find great difficulty in getting refreshing sleep there. They

are not able to sleep a sufficient number of hours, and the time for repose is frequently spent in broken sleep. Cases of insomnia in the East, due to active hyperæmia of the brain that is not relieved by rest, sleep poorly, I think, in Colorado. At least this has been my experience with some cases of this kind. Dr. Anderson, of Colorado Springs, and Dr. Sears, of Leadville, both say that cases of cerebral hyperæmia sleep well at each of the last-named places. I am inclined to believe that they have not distinguished, in their communications to me, between active and passive hyperæmia. I am led to believe that cases of passive hyperæmia of the brain, due to mental overwork, worry, loss of sleep, etc., are apparently able to obtain abundant and refreshing sleep there. I believe also that insomnia, due to any active organic brain trouble, would be made worse in Colorado. I wish to repeat that cases of insomnia, due to overwork, worry, etc., unattended by serious brain trouble, are improved by a visit to Colorado. It is very difficult to say whether medium (4,000 to 6,000 ft.) elevations, or the higher (7,000 to 11,000 ft.) are the better for cases of insomnia. We have not sufficient comparative results to enable us to determine this point yet. I never slept better than I did the eight weeks I spent in Manitou Park, at an elevation of over seven thousand feet, but then I lived in the open air all day, and slept in a tent at night. Pure air, good weather, and the amount of bright sunshine, even in winter, inviting persons to live out-doors a good portion of the day, and take more exercise than they were accustomed to do East, are important agents in enabling one to get refreshing sleep in Colorado. What lessened atmospheric pressure has to do with inducing sleep, and making it more profound, as some who reside in very high altitudes claim, has not been determined. Those who have had experience with the pneumatic cabinet and have observed the sensations felt by their patients, may be able to enlighten us on this subject.

**Irritable Nervous System.** Dr. Sears, of Leadville, in speaking of this condition, says: "It is my experience that an irritable nervous organization is rendered more so,

if there be any effect at all, which I doubt. The increased irritability, however, is only after a long-continued residence, three to five years, perhaps." He thinks those who reside in Colorado, and spend a few weeks or months at sea-level each year, do not suffer much from the irritating effects of the climate. Dr. McDonald, of Pueblo, does not think that irritable nervous systems are favorably affected by residence in Colorado, and believes that women suffer more than men, and are peculiarly susceptible to the irritating nervous effects of wind storms. Dr. Parkhill, of Denver, has related the cases of a few nervous women who were made worse by residing in Colorado, especially in the higher altitude. Dr. Strickler, of Colorado Springs, does not think the altitude of that place is productive of a nervous condition. On the contrary, Dr. Reed and other physicians of the Springs are positive that a condition of nervousness is much more common there than at low altitudes. I can account for the difference in the opinions of physicians regarding the tendency to the development of nervousness in Colorado only by the fact that there is a difference in the class of patients which each may have. Those whose practice is confined largely to the laboring class will not meet with as large a number of nervous temperaments as those who are called upon to attend the health-seekers. It has been my good fortune to be able to study a number of functional nervous troubles, both in my special line of work and in the practice of other physicians. From what I have learned from observations and inquiry, I have no hesitation in saying that the inherent nervous temperaments, not those who are nervous from malnutrition, which the climate may and does remove in many instances, are made worse by a prolonged residence in Colorado. Further, I believe, and I think that I am expressing the opinion of a number of physicians there, that many, who are not usually considered nervous, become so after a prolonged stay in Colorado. The nervousness may take the form of sleeplessness, irritable heart and tendency to passive congestions, loss of appetite, or of an



inability to concentrate the mind long on any one subject. Some complain of restlessness and irritability of temper. Persons thus affected, and contemplating making Colorado their home, should not try to overcome their sensations by prolonged and uninterrupted residence there, but they should try to spend a month or two each year at sea-level, which is almost invariably followed by an improved condition of the nervous symptoms. Dr. Reed, of Colorado Springs, informed me he had observed that child-bearing nervous women, after a prolonged stay in Colorado, recover less satisfactorily from the ordeals of the lying-in-room after the birth of the second or third child than they did after the first. The intensely bright sunshine, and the great amount of it, which is the boast of Coloradians, the dry atmosphere and the winds, it seems to me, are factors in irritating a sensitive nervous temperament. Some have tried to lay the cause at the door of lessened atmospheric pressure. This may have something to do with it, but how much it is impossible to say.

Hysteria. Dr. Strickler, of Colorado Springs, thinks we see less there than is found at low altitudes. Dr. Reed believes we see more. Dr. Tucker is of the opinion that few grave cases occur there, and Dr. Anderson says, in his experience, cases of hysteria have been few and simple. Dr. Sears, of Leadville, states: that there "it is of mild type, comparatively rare, and yields readily to treatment." Dr. McDonald, of Pueblo, believes that the number of cases of hysteria occurring there is far smaller than he witnessed in New York City. He says: that "the class in Pueblo from which we get such patients is proportionately much smaller than is found in large eastern cities." As to the effects of high altitude in causing hysteria or modifying its course, we can form no just conclusions from the infrequency of the trouble in most localities in Colorado, because, as stated by some of the physicians in their letters to me, that class, in which we find the disease most frequently, forms so small a part of the population there. Only in Colorado Springs, whose

inhabitants are mainly from the New England and Middle States, and principally from the larger cities of these do we find conditions favorable for a comparative study of the disease. Yet even there many circumstances tending to prevent the development of hysteria are present. The town is small, a social influence is felt and responded to by almost every one, and the fine weather and beautiful scenery encourage out-door life and exercise. Well-developed cases of hysteria, I am convinced, are rarer there than we find them to be proportionately in the eastern cities, and cases occurring there, it seems to me, are milder and of shorter duration. I believe more people there present nervous or illy-defined hysterical symptoms, many of which seem to be due to the direct or indirect effects of the climate, but the fully-developed cases, and severer forms of hysteria, are quite infrequent. Had we, in Colorado, all the conditions of a large eastern city favorable for the development of hysteria, I am of the opinion that the trouble would be more frequent there than it is in cities at sea-level.

Chorea. Dr. Anderson, of Colorado Springs, says: "The cases of chorea I have seen were doubtless aggravated by the altitude, and the remedy has been removal to sea-level. This is my experience in all cases dependent upon whatever cause." Dr. Strickler, of the same place, states: "I am led to believe that we see more than the usual proportion of choreic troubles. In many instances they prove rebellious to treatment, necessitating change of climate to a lower altitude, which is usually attended by benefit." Dr. McDonald, of Pueblo, writes: "I know of no points of difference in cases of chorea, neither in their course, prognosis, nor treatment, between here and at the East." Dr. Sears, who practises at an elevation of over ten thousand feet, thinks that altitude increases the frequency and severity of chorea. He has never seen a case that did not ultimately recover, but, whenever possible, he sends such patients to a lower altitude—an experiment usually followed by relief. Some of the relief he attributes to change of scene, mode of life, and differ-

ence in temperature. I have interrogated a number of physicians, in various portions of Colorado, in regard to the relative frequency, severity, and duration of chorea, and the general impression is, that the disease is more frequent there and less amenable to treatment than we usually find it at sea-level. I have, however, met with a few exceptions. The experience of some is that the disease is no more frequent and yields as readily to treatment as they found to be the case at low altitudes. Physicians giving this opinion were, for the most part from the lower elevations in Colorado. I have observed that relapses in chorea are very frequent in some of the cases that I saw there. The frequency of this, like the other forms of so-called functional nervous diseases, depends so much upon an inherent nervous temperament—a condition much less frequent in Colorado than in large eastern cities—that it is scarcely possible to make a fair comparison. I remember having seen, in consultation with Dr. Reed, a case of chorea magna which went to Colorado from New York City. The boy, who was about thirteen years old, became a great deal worse immediately on his reaching Colorado Springs. A violent paroxysm developed the first night, during which he was maniacal for several hours to an extent he had never been before. After the paroxysm, which was longer than usual, wore off, the muscular twitching and irritability of temper were severer than during the previous intervals. On leaving Colorado a few days later, he began to improve as soon as he reached a lower altitude, and showed but little disturbance when he returned to New York. Early in July of the present year, a case of chorea from Rhode Island came under my care. The boy, about thirteen years old, was attacked some time before he went to Colorado, more than two years ago. His mother, who is exceedingly nervous and subject to severe "sick headaches," says that he has not been any worse since going to Colorado, but, on the contrary, she thinks he has been a little better. The child's life in the West has been, for the most part, spent in the open air and on horseback

much of the time—a mode of life usually attended by amelioration or cure of chorea East. I have observed and treated a number of cases of the disease that developed in Colorado. It has not been my experience to find the muscular twitching worse, but the disease has been of longer duration and less influenced by treatment than I had found it in Philadelphia. When I take into consideration the open-air life, the exercise, both on foot and horseback, indulged in by children in Colorado, and compare this with life in large cities, with their close over-crowded school-rooms, illy ventilated homes, and narrow filthy streets, and yet find that chorea is more rebellious to treatment and of longer duration in the former locality than in the latter, I am forced to the conclusion that the climate of Colorado acts unfavorably on such affections of the nervous system. Colorado is too young, and the difference of life between the East and far West too great, for us yet to determine the comparative relative frequency of chorea in the latter; nevertheless, I cannot refrain from expressing the opinion that the dry air, the winds, and the elevation of most parts of Colorado are more irritating to sensitive nervous systems than the climate of low moist regions, and consequently more likely to aid in the development of chorea and similar nervous troubles.

Neuralgia. I have not been able to get many replies to my queries concerning neuralgia in Colorado.

Dr. Sears, of Leadville, writes: "The neuralgias are quite common, 'tic' being the most frequent. Sciatic, cervical, and brachio-cervical I have met. I do not think that neuralgia occurs more frequently here than in other and lower localities, perhaps not as often as in malarial districts. Malaria is not a factor in any of our diseases here, except in those persons coming from malarial districts and locating at this altitude. Neuralgias occur here as the result of 'colds,' exposure, and inherited tendency. They yield to quinia and the usual measures."

Dr. Parkhill, of Denver, narrates the case of a strong healthy woman, who went to Colorado, and was first

attacked by irritability of the bladder, which was followed by facial and intercostal neuralgia, coccydynia, and a condition of general nervousness, with the loss of flesh and strength. He mentions the case of a nervous woman of Cleveland, Ohio. She went to a high mountainous region of Colorado, and was almost immediately attacked with a severe sciatica which did not improve until she went to a much lower altitude (Denver), and did not cease entirely until she returned to her home in Ohio. Dr. Parkhill thinks the altitude in these cases had much to do with the neuralgic troubles, and expresses his belief in neurotic individuals being unpleasantly affected by the higher altitudes of Colorado. He attributes much to the influence of diminished atmospheric pressure. From my own experience, and from what I have learned from other physicians, I cannot agree with Dr. Parkhill in thinking that nervous persons are especially prone to attacks of neuralgia on going to high mountainous regions. I have seen a number of nervous women who had gone to Colorado from the eastern States, but in none have I known of a case of neuralgia, in whom attacks of the disease had not occurred while they were living at sea-level. Dr. Strickler, of Colorado Springs, thinks that neuralgia is less frequent and less severe there than at low altitudes. During my stay there of three years, I have seen much less general peripheral neuralgia than I had met with in the same number of persons while practising in Philadelphia. I think one reason why neuralgia is not more frequent in Colorado is because of the slight amount of malaria there. Pleurodynia and various chest pains are more common in consumptive patients there than are found in the eastern States. I have seen two patients suffering from intercostal neuralgia with herpetic eruptions. One case occurred in a strong young man, a nurse who had been fatigued by his night watchings. It was light and of short duration. The second case was in a consumptive man. The attack was severe and persisted for months. I have known of four cases of migraine or "sick headache." They were all subject to the disease before going to Colorado. The

trouble was apparently a little better, for a short time, after making their homes there, but it soon became as severe as it had been formerly, and, I think, of late the attacks have increased in frequency, although not in severity. I have been able to hear of but one case of migraine that developed at Colorado Springs, and that followed an injury to the head.

Dr. C. H. Hughes, of St. Louis, in speaking of neuritis plantaris, says: "I have seen it follow upon a residence in the high altitudes of Colorado, and after an attack of the so-called mountain fever of that region." (*Alienist and Neurologist*, April, 1887.)

Epilepsy. Dr. Strickler, of Colorado Springs, states: "I am confident that epilepsy is rarely, if ever, benefited by a resort to this altitude; on the contrary, it is almost always aggravated. It quite frequently originates here, and proves rebellious to treatment. I think more than the usual proportion of cases are of the lighter form, known as *petit mal*."

Dr. Anderson, of the same place, writes me: "I have seen four cases of epilepsy. One, due to syphilis, yielded to proper treatment; the other three were imported cases, and were neither benefited nor aggravated by residence here."

Dr. Sears, of Leadville, remarks: "I have had very little epilepsy to treat here, and in a residence of seven years have treated only three cases; two *grand* and one *petit mal*. The two subject to the severer form of disease were hard drinkers. I was unable to obtain the history of the case of *petit mal*, as he passed from under my observation shortly after I first saw him. I do not think that epilepsy occurs here primarily, and am quite certain that its attacks are much modified by the climate in those suffering from the disease, especially when suitable treatment is instituted."

Dr. Tucker, of Colorado Springs, during an active practice of some six or seven years there, has known of only one or two cases of epilepsy that developed in Colorado. I have no personal knowledge of a single case of epilepsy

that originated there, and have only seen two or three cases during a residence of three years in Colorado Springs. These did not seem to be materially influenced by the climate, but the length of their stay there was too short to enable one to form definite conclusions. I am inclined to believe that cases of epilepsy associated with extreme anæmia might be benefited by a residence of a few months in Colorado, provided the patient avoided excitement and exercise. On the other hand, traumatic epilepsy, and epilepsy complicated by active hyperæmia of the brain would probably be aggravated by the climate.

Insanity. Dr. Strickler, of Colorado Springs, says: "As I am not familiar with the statistics of insanity, I am not prepared to speak of its relative frequency. I think we rarely see here cases of mental derangement with delusions of exaltation. Nearly all the cases that I have seen have been of the melancholic type from the beginning. Such cases are, I think, comparatively frequent."

Dr. Sears, of Leadville, states: "I have known of six or eight cases of insanity that have occurred here during the past eighteen months or two years, and perhaps of a dozen during the seven years that I have resided at Leadville. I cannot say how much the altitude has had to do, either as a cause or as a modifying influence of the disease, as many of the cases occurred in improvident and debauched persons. In three instances, at least, insanity developed in persons who were financially comfortable, and were in apparently good health just before the onset of the alienation. I am convinced, however, that there is more insanity here than is usual in like communities at low altitudes, but there are other features than altitude which must be taken into consideration in studying the cause and frequency of insanity of this region. Most of the cases have occurred in gamblers or miners who were hard drinkers. The worries and uncertainties of speculation, mining, and the gambling table, together with insufficient sleep, poor food, and exposure to the severe winter weather of this place, must have much to do in causing insanity."

Dr. Macdonald, of Pueblo, has not been able to perceive any special causative or modifying influence of insanity in the climate or altitude of that place, but says: "There are certain forms of nervous trouble, notably neurasthenia with a tendency to depression of spirits, and melancholia that are promptly and markedly benefited by our sunlight and atmosphere."

The writer's experience with insanity in Colorado has been rather large for a place the size of Colorado Springs, but as all cases that occur in El Paso County are taken to the county seat, and adjudged insane by a jury of six men, before they can be sent to the State Insane Asylum, I have been fortunate enough to see and examine a large proportion of the cases that have developed there or come into that county during the time I have resided at Colorado Springs. I have records of eleven cases that I have seen there. The occupation of five was ranch life, of two laboring work, one was a gardener, one a carpenter, one a book-keeper, and one a prostitute. Ten are males, and one is a female. The cause of insanity in four was alcohol, in two trouble, in two nothing was found beyond heredity, in two no cause was found, and in one the alienation came on after an attack of typhoid fever. Of the eleven cases, four were cases of paranoia, three dementia, one melancholia, one parietic dementia, one mania, and one circular insanity. So far only one of these cases has been declared "cured," and the recovery in this is extremely doubtful.

It is the impression with many physicians in Colorado that insanity is more frequent there in proportion to the population than it is in the Eastern States. This may be true in some mining districts with great elevation like Leadville, but it does not hold good throughout the State. According to the United States census for 1880, there were in the United States one insane person to 297 of the population, and in Colorado, which had the least number of insane with two (Wyoming and Arizona) exceptions of all the States and Territories, there was only one insane person to 1,104 of her population (Pepper's



"System of Medicine," vol. V., p. 112).<sup>1</sup> Colorado is too young to have an insane population in proportion to the older eastern States. I believe that insanity is rapidly on the increase there. I have known of a few cases of insanity that have developed in Colorado and been improved by being sent to a low altitude. I believe that the very high altitude of some portions of Colorado, and the habits and mode of life of many of her citizens, are productive of brain degeneration and insanity. Ranch life, especially among sheep herders, which, by the way, is one of the most lonely and solitary that can be imagined, gives a large per cent of the cases of insanity. The report, for 1886, of the Superintendent of the Colorado State Insane Asylum, gives intemperance as the cause of insanity in thirty-seven of one hundred and ninety-one patients admitted.

In this connection, I wish to report a case of mental confusion or amnesia, caused, apparently, by high altitude. It is the only case of the kind of which I have any personal knowledge. An intelligent young man, a tutor, in excellent health, started from Manitou early one morning, in June of the present year, to go on horseback, to the summit of Pike's Peak. The distance is about twelve miles. He had eaten a fair breakfast, but took no stimulants that day, neither before nor during the trip. He accomplished the ascent in a few hours, in company with several others. The party remained on the peak about two hours before beginning the descent of the mountain. Nothing peculiar was noticed in the young man until he had descended about two thousand feet, when some of the party observed his strange remarks

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<sup>1</sup> One reason why the proportion of the insane of Colorado is so small is because many who become insane there are sent to eastern asylums where the altitude is lower, so that few of Colorado's insane go to her State asylum, except those who have no means to pay their expenses for treatment. The same statement holds true of the other States and Territories among the Rocky mountains. There is a popular impression that high altitudes injuriously affect insane persons and, in consequence, every effort is made by the relatives and friends of the insane to get them at sea-level if possible.

and absent-minded condition. It was found, on inquiry, that he had forgotten nearly everything that had occurred during the day. When he reached Manitou, late in the afternoon, he did not remember at what hotel he had been stopping. He had paid for the hire of his horse, and his guide for his services, in the morning before starting, but on returning had forgotten all about it. When he reached his room in his hotel, he could not remember what he had done with his horse, and started to look for him. He remained in this condition about thirty-six hours. I fortunately had an opportunity to interview him a few days after the strange occurrence. At the time of my conversation with him, he said that he then remembered every incident of the day's journey, of which he was oblivious the day of the ascent to the peak. He told me that he was not conscious at the time anything was wrong with his memory, but was conscious of saying foolish things to which he could not help giving expression. He could now recall his dazed condition, loss of memory, and the cause of the laughter which he provoked among his party. He says that he had ascended several mountains as high, and some higher than Pike's Peak, but never before had he had a similar experience from mountain climbing.

For more than a year I have been trying to collect the experiences of those who have ascended high mountains, with reference especially to nervous effects produced at great elevations. I have found almost nothing written on the subject. Mr. Ralph Copeland, in "Copernicus," vol. III., 1883, in an account of his astronomical and meteorological observations at high elevations in the Andes Mountains of South America, mentions the fact that, at altitudes of more than 14,000 feet, he found blacksmith shops supplied with heavy sledges which he supposed the natives wielded with as much ease and convenience as is done at sea-level. However, this is not positive, as it appears that he made no inquiries concerning the matter. Of his own experience, he gives almost nothing. From his statements it seems he experienced no unpleasant sensation at an altitude of 18,000

feet, except a slight oppression in breathing which he could relieve by a few deep inspirations. Mr. Graham, and his companions who had been guides in the mountains of Switzerland, have experienced no bad effects from mountain climbing in Asia at an altitude of 23,000 feet.

In July of the present year, a dentist of Boston, member of a mountain climbing club, went to Este's Park, Colorado, an elevation between 8,000 and 9,000 feet, and we may suppose exercised more or less. Whether he ascended Long's Peak, I was unable to learn. At any rate, at the end of a week or two, he experienced a sensation of intense heat, felt, as he expressed it, as though he were in a furnace, and began to lose flesh and strength rapidly. He left that altitude. His subsequent history I have been unable to learn, as this occurred only a short time before I left Colorado for Philadelphia.

Most of the information which I have been able to gather concerning the effects of high altitudes has been from tourists and others, who for the most part, unaccustomed to great elevations, have made the ascent of Pike's Peak. Many have told me that they observed nothing except a feeling of exhilaration. I think the majority of persons who go to great elevations feel the stimulating effects of the rarefied atmosphere, although a number go to the top of the peak, eat, drink and smoke, with the same impunity that they do at sea-level. Some, however, are troubled with a feeling of great lassitude which remains as long as they stay at the summit of the peak, but begins to lessen as they accomplish the descent of the mountain. I have known of one or two persons who have fainted on reaching the top of the peak. Most, if not all, of these who experience a stimulating effect at great elevations, also have a sensation which they describe as being like sea-sickness. I cannot say that none of those who have a sensation of lassitude at great elevations experience a feeling of nausea, but so far I have not met with a case that suffered from nausea and lassitude from great elevations.

Professor Pickering, of the Astronomical Department

of the University of Harvard, his brother, and several assistants, paid by the Henry Draper Memorial Fund, are engaged in making observations in astronomy and meteorology, at the higher elevations at the Rocky Mountains. Most of the party are trained in making accurate observations in physics, and are enthusiastic in their work. Professor Pickering has enlarged his field of work, by making observations on the effects of high altitude on man. As these observations will be carried on for a number of years, both in the Rocky Mountains and in the Andes, at elevations varying from 10,000 to 20,000 feet, we may fairly hope for interesting results.

**Inflammatory Lesions of the Brain and Cord.** Dr. Sears, of Leadville, says: "These lesions, especially those of the cord are rare."

Dr. McDonald, of Pueblo, states: "Inflammatory lesions of the brain and cord have not been sufficiently frequent to enable me to form any conclusions as to the effects of climate upon them."

Dr. Anderson, of Colorado Springs, remarks: "The only lesion of the brain with which I have had any experience here, has been softening, and I would say from experience that long residence in high altitudes is one of the most prolific sources of this affection. A number of cases in 'old timers' have come under my observation, and have proved fatal."

Dr. Strickler, of the last-named place, observes: "Contrary to what would be supposed, inflammatory lesions of the brain and cord I believe to be comparatively infrequent. Such diseases are very rare in infancy. I cannot call to mind more than three cases of what I diagnosed tubercular meningitis, during the seventeen years that I have been in practice here. According to my experience, all acute and chronic inflammatory troubles of the central nervous system are comparatively rarely met with at this place."

Dr. Reed, of the same place, thinks he has met with tubercular meningitis more frequently in Colorado Springs than he did in the same number of children, either

in Philadelphia or Michigan. Drs. Tucker and Hart, of Colorado Springs, speak of having seen a number of cases.

During my stay there of three years, I have seen three fatal cases of tubercular meningitis. The duration of this disease seems to be a little shorter in Colorado than at lower altitudes. One case that I saw, proved fatal in about thirty-six hours from the time the patient, a consumptive young lady, took to her bed. Children, whose parents have died or are suffering from consumption, form so large a portion of the youthful population of Colorado Springs that I am surprised that tubercular meningitis is not more frequent than it is. It may be that the open-air life led by the children, and the bracing effects of the atmosphere, together with cool nights, even in mid-summer, insuring refreshing sleep, enable the issue of consumptive parents to overcome the tendency to the development of the disease. Certainly this seems to be the case with reference to the development of tuberculosis of the lungs, in children that are born and reside in Colorado. One reason, I think, why idiopathic meningitis is almost unknown in many parts of Colorado, and why tubercular meningitis is not more frequent, seeing there are so many children whose parents are consumptive, is because of the great dryness of the atmosphere, which permits of rapid evaporation of the perspiration.

I have never seen a case of infantile paralysis in Colorado. It is evidently exceedingly rare. On inquiring of several physicians of Colorado Springs, I find they have never seen a case there. I have not met with or heard of a case of spinal-cord disease of any form in a child at the Springs. I have seen three cases of paralysis agitans, four or five of posterior spinal sclerosis, one or two of multiple sclerosis of brain and cord, and one of Landry's disease. So far as I was able to ascertain, they had all, except the case of Landry's disease, developed at low elevations. Their duration and prognosis appear to be about the same as found to be further East. In some cases nutrition has so improved, as, for a time, apparently to arrest the

progress of the trouble. Dr. Solby thinks he has seen temporary good effects produced by a residence in Colorado on chronic inflammatory disease of the cord. Dr. Anderson is firmly of the opinion that premature brain degeneration, or chronic softening, is quite frequent in Colorado, especially in those who have spent much time at the higher altitudes. This is the popular impression, and, in this respect, Dr. Tucker's experience accords with Dr. Anderson's. The writer's limited experience with these cases there does not enable him to express an opinion. He has seen no cases in Colorado in which he has been able to make a diagnosis of tumor or abscess of the brain.

Apoplexy. Dr. Strickler, of Colorado Springs, says : " I have not had a case of hemorrhagic apoplexy in this country. This strikes me as very singular, and what is still more so, I cannot call to mind any case that has occurred in the practice of any other physician here. Doubtless one reason for this lies in the fact that the country is comparatively newly settled, so that but few aged persons are among us. Still the absence of this form of apoplexy is quite phenomenal. A number of cases of embolism of the middle meningeal arteries, and consequent softening and death, have come under my observation."

Dr. Anderson, of the same place, states : " I have never seen a case of genuine hemorrhagic apoplexy here."

Dr. Sears, of Leadville, writes : " I have seen three cases of apoplexy during the last year. None were fatal. All the cases occurred in drinkers ; two were sots, one an occasional tippler. The two former are hemiplegic, the latter suffers from amnesiac aphasia. I do not think that altitude exerts much influence as a cause of apoplexy."

In May of the present year, I saw a case of hemorrhagic apoplexy at the Springs. About the same time another case of apoplexy occurred there. From what I could learn from the symptoms, I presume the latter was embolic. It proved fatal after repeated attacks, but no post-mortem examination was made. I see no reason why

hemorrhagic apoplexy should not be as frequent in Colorado, in those advanced in life, as we find it at sea-level. The question is still unsettled, and it will require a few years' further observations to determine the comparative frequency. In 1886, I saw, in consultation with Dr. Tucker, a fatal case of what I diagnosed thrombosis of the basilar artery. It is the impression of most of the physicians of Colorado Springs that disease of the cerebral arteries is not infrequent in Colorado. The cases of chronic degeneration of the brain which, according to some physicians, occur so frequently there, are, in my opinion, softening due to embolism or thrombosis of some of the cerebral arteries.

Sunstroke, I believe, is unknown in most parts of Colorado. It is said never to have occurred there. Its absence is accounted for by the active peripheral capillary circulation and by the free evaporation from the surface of the body, the former kept up by the rarefied condition of the atmosphere, and the latter is caused by its dryness.

I have known of no cases of paralysis, paresis, anæsthesia, hyperæsthesia, paræsthesia or numbness, coming on in Colorado, as the result of high altitudes.

EFFECTS OF THE USE OF ALCOHOL, TOBACCO, COFFEE AND TEA IN HIGH AND DRY MOUNTAINOUS DISTRICTS. Alcohol is thought, by some, to be more rapid in its action and more transient in its effects in Colorado than at sea-level (Solby). I doubt whether this is so to a sufficient extent to make any practical difference in its effects on man. It is the general impression, and one which many of the physicians of Colorado share to a certain degree, that alcohol cannot be taken in large quantities and continuously there, without being followed by unpleasant symptoms more quickly than we find is the case after its use in the same manner at lower altitudes, especially where the climate is damp. We have seen that persons suffer more from functional nervous troubles in Colorado than they do in a different climate. The nervous system, in persons

who reside there long, generally seems to be more irritable, more easily excited, and quickly to resent an excitant.

Smoking, especially during the latter part of the day, is a great cause of sleeplessness in Colorado. I think but few find they can smoke as freely there without unpleasant symptoms as they can at sea-level. Irritable heart and a condition of nervousness follow the use of tobacco at high and dry mountainous places more quickly than at low altitudes where the atmosphere is damp. I believe that persons should be more cautious in the use of alcohol, tobacco, tea, coffee and all nerve stimulants and sedatives in Colorado than they need be along the sea coast.



## MICROSCOPICAL STUDIES IN A CASE OF PSEUDOHYPERTROPHIC PARALYSIS.<sup>1</sup>

By DR. GEO. W. JACOBY,

PHYSICIAN TO THE CLASS FOR NERVOUS DISEASES AT THE GERMAN DISPENSARY OF THE  
CITY OF NEW YORK.

NOTWITHSTANDING that a great amount of attention has during the last few years been devoted to the subject of primary affections of the muscles, and that a great deal has been accomplished towards the correct classification of the various forms encountered, nevertheless our ideas in regard to the processes and anatomical changes which occur in these affected muscles are still in a condition of undesirable confusion, and it seems to me that, before we can clear up the clouds from the clinical field of vision, we must do so from the pathological one. An affection which we know to be a primary myopathy, for it is well settled that here the central nervous system and the anterior nerve roots are not affected, is the form known as pseudohypertrophic paralysis. But what the histological changes are, as they occur in this affection, is not yet definitely decided. Notwithstanding the numerous examinations of muscles which have been made, there does not appear to be an entire uniformity of opinion as regards the anatomical changes which take place, and it is therefore very probable that these changes vary in different stages of the affection. For this reason every careful and impartial observation must be of value. It is in this spirit that I take the liberty of presenting the results of the microscopical examinations in the following case.

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<sup>1</sup> Read before the Amer. Neurol. Association, Long Branch, N. J., July 20th, 1887.

The pieces of muscle examined were excised from a patient, whose history is a classical one of pseudohypertrophy. The case was a typical one, so that the description can be made very short.

*Sept. 30th, 1885.* H. P., æt. 15. Family history unimportant. No heredity. Mother had seven children, of which two died. One of these was paralyzed, probably a diphtheritic paralysis. The others, with exception of the patient, were all well. He was brought up at the breast. Began teething at eight months. At thirteen months could walk. At eighteen months he had convulsions, which repeated themselves during four days. Then he was unable to walk for four weeks, but subsequently he walked as well as ever. His mother says he always had "crooked legs." He went to school and did not show any backwardness. At age of 9 years he had a severe fall, which necessitated his confinement to the house for eight weeks. From this time the mother first noticed some awkwardness in his gait. At the age of 12 years it was noticed that mounting stairs afforded him particular difficulty. His condition gradually grew worse and worse; he fell easily and any slight jar was sufficient to make him lose his balance.

*Status Præsens.* Upon examination the usual symptoms of pseudohypertrophic paralysis are found, such as, letting himself fall into the chair in sitting down; placing his hand on his knees and drawing himself up when arising; difficulty in mounting stairs, etc. Examination shows hypertrophy to an enormous extent of both vasti externi, of both calves, of the glutei, infraspinati, deltoidei, biceps, triceps, and costal portions of the pectoralis major. No atrophy of any muscles could be made out.

Pat. tendon reflexes absent.

Hypertrophied muscles show reduced excitability to both currents. Otherwise nothing abnormal to be found.

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Two pieces of muscle, each of about the size of a bean, were removed from the quadriceps femoris of the left leg. The muscle was placed at once in a one-half per cent solu-

tion of chromic acid, and after a sufficient amount of hardening had taken place it was imbedded in celloidin and transverse and longitudinal sections made. These were then stained with an ammoniacal solution of carmine and mounted in glycerin. Transverse sections examined by low power (200 diam.) reveal a striking variation from normal muscle in the number of muscle fibres as arranged into groups by the perimysium internum. Whereas a normal quadriceps femoris shows between thirty and fifty muscle fibres in a bundle, the diseased muscle averages about

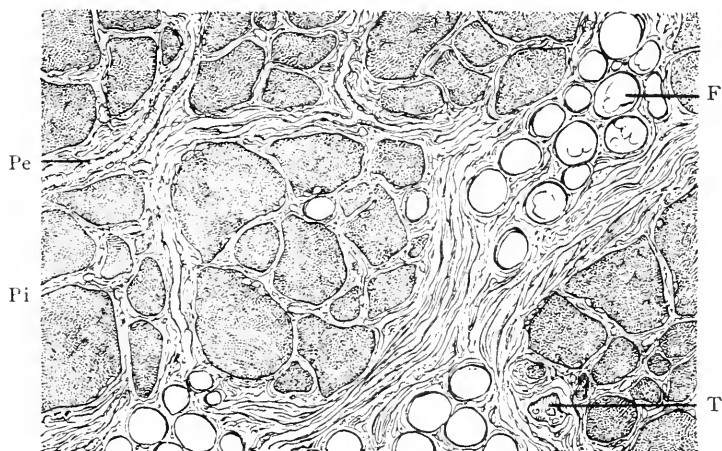


FIG. 1.—Quadriceps femoris from pseudo-hypertrophic muscle.  $\times 300$ . Transverse section.

*Pe*, Perimysium externum (carrying arteries and veins). *Pi*, Perimysium internum (carrying capillaries). *T*, Tendonlike formations, formed from previous muscle-fibre. *F*, Fat globules.

one-half that number. At the same time it is conspicuous that the size of the single muscle fibres is on an average smaller than normal, but that there are a number of fibres of normal size present. Hypertrophic fibres were sought for carefully in all my specimens, but in vain. Although variations in the thickness of the fibres have been noted by most all observers, some finding atrophic, normal, and hypertrophic, others finding only normal and hypertrophic ones, the majority of later observers, particularly

Schultze and Erb, claim that hypertrophic fibres are always present. Schultze goes so far as to make the principal microscopical differentiation between neurotic and non-neurotic atrophies dependent upon the hypertrophy or non-hypertrophy of muscle fibres. But he says that this hypertrophy may be absent in pseudohypertrophy and analogous affections, if at the time of the examination general marasmus existed. This certainly was not the case in my patient. It must, however, be acknowledged that perhaps a more extended examination of other muscles might have revealed their presence.

In contradistinction to this atrophy of the muscle fibres, the connective tissue constituting the external and internal perimysium is decidedly augmented, to such a degree in fact that its bulk is twice and in some places three times that found in normal muscle.

The perimysium internum in a normal muscle is approximately uniformly distributed between the single muscle fibres, being of sufficient breadth only for carrying a capillary blood-vessel. In the diseased muscle, such a limitation of breadth is but exceptional, and the average bulk is sufficient for carrying several capillaries, in some places even attaining the size of the perimysium externum of normal muscles.

Thus it often becomes impossible to differentiate between internal and external perimysium; a small group of muscles fibres (as in one specimen ten) being surrounded by a layer of connective tissue fully as broad as the normal perimysium externum.

This latter, in the diseased muscle, is conspicuous by the presence of arteries and veins, and of two kinds of bundles of fibrous connective tissue, the one being the delicate fibrous tissue as seen in normal muscle, and the second being broad and dense, resembling the fibrous connective tissue of aponeuroses and fasciæ.

Besides this, the perimysium externum is, in many places, crowded with or replaced by fat tissue.

The general impression gained at the first glance is that the number of muscle-fibres is reduced, the mass of con-

nective tissue, on the contrary, being decidedly augmented. These facts correspond to reports of all other observers, since first described by Duchenne, and four years later by Griesinger, and form the basis of the name pseudohypertrophy.

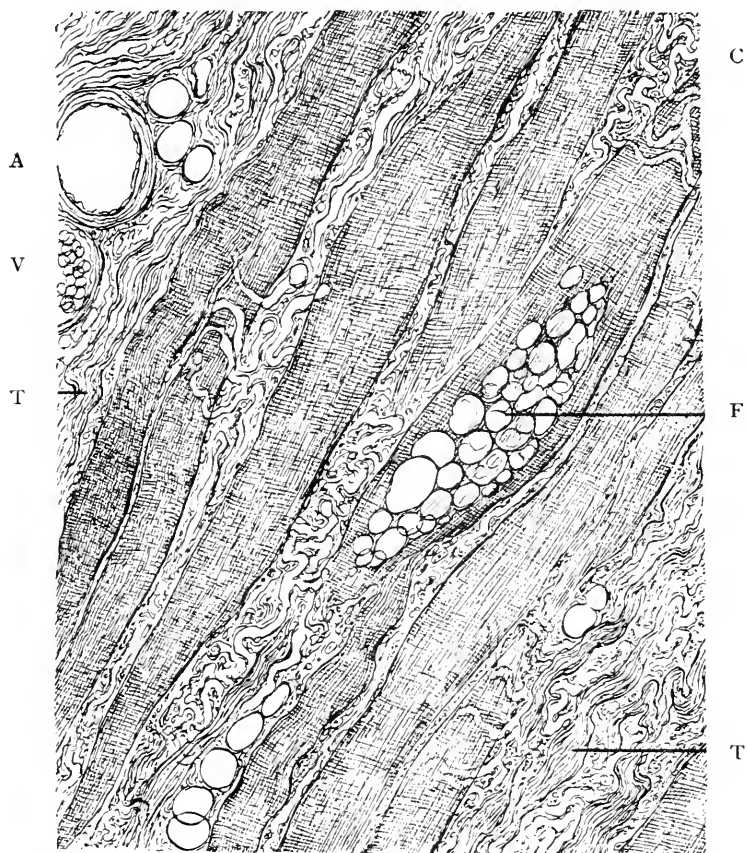


FIG. 2.—Quadriceps femoris from pseudo-hypertrophy.  $\times 300$ . Longitudinal section. A, Artery. V, Vein. C, Capillaries. TT, Tendon-like formations of fibrous connective tissue arisen from previous muscle-fibre. F, Fat globules in muscle-fibres.

The question which, after such a view, forces itself upon the mind is, Whence comes this augmented connective tissue?

This question is, to my knowledge, not answered satisfactorily by any of the writers upon this subject. They speak of chronic interstitial connective-tissue hyperplasia (Friedreich, Erb), proliferation of connective tissue (Buss), and Schultze describes the microscopical picture as being one in which the muscles are partly replaced by normal fat and normal connective tissue, while the fibres themselves are partly in a state of simple atrophy; and in another place, he speaks of the appearance of large connective-tissue tracts in the muscles. Thus we see no satisfactory explanation is given as to their origin. The fact that the muscle-fibres are decreased in number as well as in size is in itself suggestive of the source of the augmented connective tissue. And, actually, all my specimens go far to prove that the connective tissue has grown, to a great extent at least, at the expense of the contractile substance.

Not only are the nuclei of the muscle-fibres augmented, and here and there arranged in chains, but in many places the peripheral portions of the fibres are transformed into clusters of medullary or inflammatory corpuscles. These clusters are, as a rule, seen along one side of the muscle-fibres only, very exceptionally on both sides. Not infrequently the muscle-fibre, in a *longitudinal section*, appears to be split into smaller pieces by chains of medullary corpuscles, the presence of which leads to a division of the original muscle-fibre into two or more sections. This process of splitting up or dichotomous division is well known since the descriptions of Cohnheim and Knoll, and has been uniformly observed by the latest observers (Erb, Schultze, Buss). Taking these divisions into consideration, we can understand why, in a transverse section of the affected muscle, the number of narrow fibres is so much in excess of those visible in normal muscle, where their presence becomes explicable by the spindle shape of such individual muscle-fibre. Examined with high power, the fact becomes established that the medullary corpuscles have originated from the muscle substance itself. Bay-like, crescentic, or elongated excavations of

the latter are found filled up with medullary corpuscles, and either sharply defined towards the muscle, or gradually blending with it. In the latter case, we observe a breaking up of the muscle-tissue into bodies which are at first homogeneous and later granular and nucleated, and which occupy only the periphery of the muscle-fibre in certain cases, and in others are seen penetrating its entire substance.

In many places, we can observe a gradual transition of the medullary corpuscles into spindles, and through these into fibrous connective tissue. However, not only the delicate, but also the coarse fibrous connective tissue seems to be an offspring of muscle-tissue. This coarse tissue especially is not infrequently seen in tracts of the breadth of original muscle-fibres, and often bordered on either side by the delicate variety. This fact is suggestive, in a measure at any rate, of the origin of the coarse bundles from an entire previous muscle-fibre.

The newly-formed connective tissue itself is seen, in various places, to be the seat of an inflammatory infiltration, and in this condition exhibits the characteristic adenoid or myxomatous structure, that is, clusters of globular shining corpuscles imbedded in the meshes of a delicate fibrous network.

The muscle-fibres themselves are conspicuous by their longitudinal striation, which signifies a prevailingly longitudinal arrangement of the sarcous elements. These are invariably extremely minute in size. Their arrangement into groups, due to the embryonal sarcoplasts, is not, very marked; hence, also in transverse section of the fibres the traces of Cohnheim's fields are rather indistinct.

This fact would, according to my judgment, indicate the existence of a malformation of the muscle-tissue existing from the earliest stages of its development.

In some muscle-fibres, the centre is occupied by clusters of coarse, shining granules, likewise a pathological feature, indicative either of a reformation or of a beginning inflammation in the centre of the fibre.

Exceptionally there are present narrow muscle-fibres

in which all striations are lacking, which have a marked gloss and are deeply stained with carmine.

This is the change which, by previous observers, has been described as colloid or hyaline degeneration of the muscle fibre.

The fat tissue, as already mentioned, is present in a

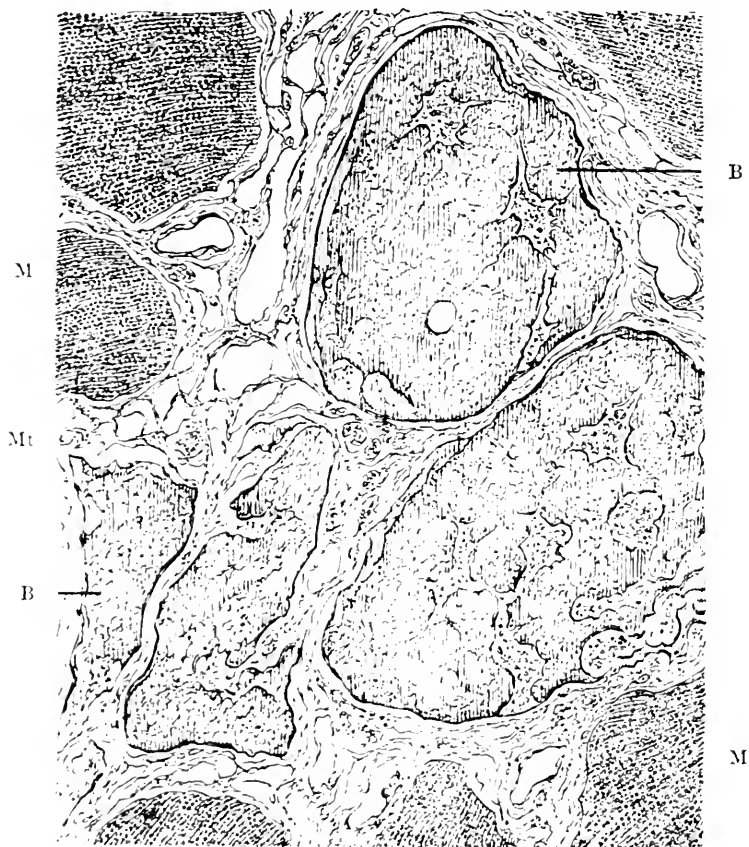


FIG. 3.—Quadriceps femoris from pseudo hypertrophy.  $\times 500$ . Transverse section. *MM*. Muscle-fibres, cut obliquely. *Mt*. Myxomatous tissue of perimysium. *BB*. Transverse sections of bundles of tendinous tissue, resembling fields of hyaline cartilage. (In a granular, partly homogeneous basis substance are imbedded branching protoplasmic bodies, in part interconnected.)

large quantity in the newly-formed connective tissue. Exceptionally, however, fat globules are seen in the interior of the muscle-fibre. That the fat globules are ac-



tually in the centre of the fibre, and not on the surface, can easily be ascertained in longitudinal sections where the clusters of fat globules are seen to be bordered on all sides by striped muscle-tissue. Obviously, the inflammatory change has in this instance led to a new formation of myxomatous tissue, whose meshes then became the seat of fatty deposit.

The amount of fat may vary from the presence of a few globules to that of large clusters, which latter lead to a corresponding increase of the diameter of the muscle-fibre at that point. These observations correspond entirely with those of other writers.

Peculiar interest is attached to formations which are seen in the perimysium, and which, at the first view, convey the impression of fibrous connective tissue devoid of striation. These formations are conspicuous by their high refracting power, their composition of small angular pieces, their wavy contour, and their deep staining with carmine.

In some places they are so numerous that the muscle-fibres are completely replaced by them, it being noticeable, however, that they correspond in breadth and general configuration with these fibres.

High powers (5-600 diam.) show that these tracts are composed of bluntly angular lumps between which interstices of various sizes are visible, filled with protoplasm. The angular fields again are made up of smaller pieces indistinctly separated from each other, and corresponding in size with the medullary corpuscles.

Cross sections of these tracts are at once recognizable by their high refracting power and their deep carmine stain.

The protoplasmic bodies visible in the interior of these tracts are sometimes narrow and branching, like tendon corpuscles in transverse section, sometimes, on the contrary, they are oblong or slightly irregular, without larger offshoots, closely resembling the appearance of cartilage corpuscles. There cannot be the slightest doubt as to the

origin of such tracts from previous muscle-fibres (Figs. 4 and 5).

*I have, in many places, seen transverse sections of these tracts surrounded by a delicate perimysium, a portion of the tracts still exhibiting all the characteristic features of*

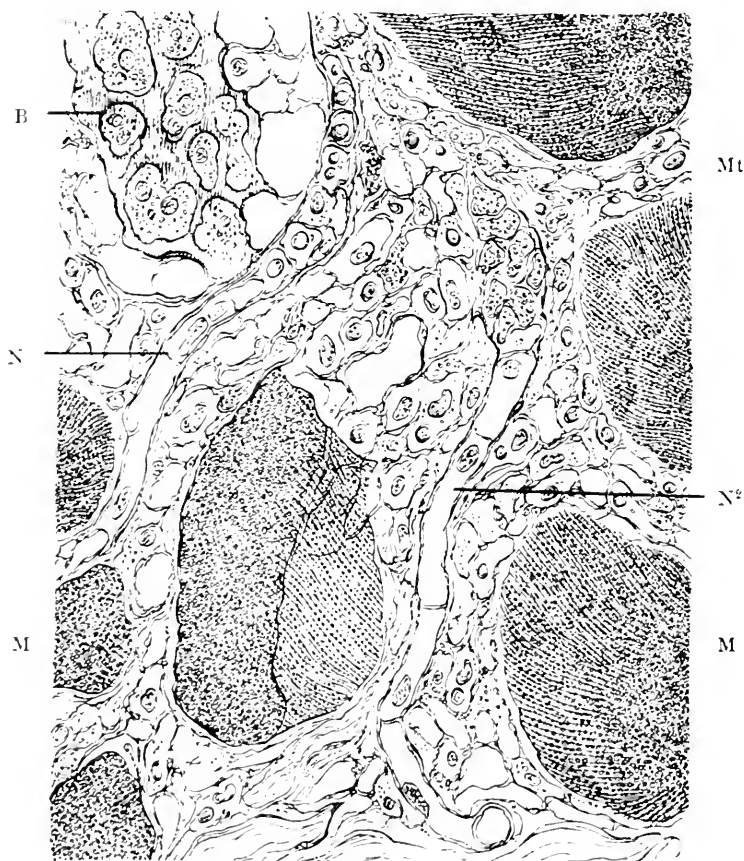


FIG. 4.—Quadriceps femoris from pseudo-hypertrophy.  $\times 500$ . Transverse section.  
(Nerves.)

$N^1$ , Medullated nerve-fibre terminating in a hilly formation composed of myxomatous tissue, possibly a muscle-plate.  $N^2$ , Medullated nerve-fibre terminating in a cluster of inflammatory corpuscles. (Neuritis.)

$M, M$ , Muscle-fibres in transverse and oblique sections.  $Mt$ , Myxomatous tissue of perimysium exhibiting all the features of granulation tissue.  $B$ , Tendon or cartilage-like bundle with numerous and large protoplasmic bodies.

striped muscle-tissue, whereas the surrounding mass was made up of medullary corpuscles more or less advanced in their transformation to basis substance. As regards the nature of these tracts which, at least to my knowledge, represent a novel feature, not only in the study of pseudo-hypertrophy, but in the pathology of muscle-tissue in general, I can say that some of them have the appearance of coarse aponeurotic connective tissue, others that of hyaline cartilage. The morbid process has led to a transmutation of the muscle-fibres into varieties of connective tissue, either aponeurotic or cartilaginous. Obviously this change has not taken place through an immediate transformation, but through the intermediate stage of medullary tissue which, in its turn, has been infiltrated with a chondrogenous basis substance, and thus has led to such a peculiar result.

The vascularizations of the perimysium, that is, the delicate connective tissue only, is, in some places, rich, and evidently surpassing that of normal muscle.

The capillaries form plexus around the muscle-fibres, and are conspicuous by their large calibres and their tortuous course.

I do not doubt that many of these capillaries are newly formed.

I have traced a number of medullated nerve-fibres, which in part were unchanged, and terminated in the usual motor hill on the surface of the muscle-fibre. Other nerves, on the contrary, exhibited augmented nuclei in Schwann's sheath, or they were broken up into rows of medullary or inflammatory corpuscles, or they were lost in fibrous connective tissue, identical with that of the perimysium. The general number of medullated nerve-fibres is, in all of my specimens, noticeably decreased.

If, now, from the results of this examination I may be allowed to draw conclusions without, at the same time, generalizing, I shall, in a measure, differ with other observers.

I am convinced that in my patient the disease is essen-

tially a chronic inflammation invading *both the perimysium and the muscle-tissue*. It is impossible to say what the cause of this process may be, unless it is to be sought for in a congenital malformation of the muscle-tissue itself. Such a malformation is at least indicated by the strikingly small size of the sarcous elements, as we are accustomed to see them in the earliest stages of embryonal development. However this may be, the pathological process consists in a gradual reduction of the muscle-fibres into medullary or inflammatory corpuscles, which in time go to build partly fibrous, partly cartilaginous, and partly fat connective tissue. The process, which is extremely slow, gradually leads to an augmentation of myxomatous or other varieties of connective tissue at the expense of the muscle-tissue. I may here say that I cannot agree with Gowers and Buss, that the proliferation of connective tissue is the primary, and the disease of the muscular tissue the secondary process. Either the reverse of this is true, or the process occurs simultaneously both in the muscle and in the perimysium.

I do not hesitate to place the entire process, as seen in my specimens, in the same category with the process termed *myositis ossificans progressiva*, and had I to describe it in a few words would call it a *myositis progressiva hyperplastica*.

Of the various investigators of the subject during the last few years, Jurgens is the only one who also considers the process a myositis; but he looks upon it as an interstitial myositis alone, and says that he is not prepared to give an opinion regarding the changes in the muscle-fibres themselves.

The more frequently careful microscopical examinations are made of the muscular tissue in the various dystrophixæ, the more are we obliged to come to the conclusion that, either from a microscopical examination alone we cannot always make a distinct diagnosis between the various affections, or that they are, as is largely admitted to-day, all varieties of one and the same process, which imperceptibly blend with one another.

What particularly impressed me in this connection was that my examination differed so decidedly from descriptions of pseudohypertrophic muscles as given by others, yet, notwithstanding that the case was clinically a perfectly clear one, agreed almost entirely with the description given by Friedreich of the muscle from a case of progressive muscular atrophy (case 10, page 37), which affection, as is well known, he classed as a primary myopathy, and considered the process to consist of a chronic progressive myositis.

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## Clinical Cases.

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### NOTE ON A CASE OF INSANITY OF DOUBT.<sup>1</sup>

By JAMES HENDRIE LLOYD. A.M., M.D.,

INSTRUCTOR IN ELECTRO-THERAPEUTICS IN THE UNIVERSITY OF PENNSYLVANIA.

The form of mental disease, which has been described by some French writers as the insanity of doubt, has presented itself to my notice in a number of cases, to one of which I wish briefly to call your attention. I am not certain at all that you will find the details either novel or complete, in fact I know that they have been observable if not observed, just as I also know that I have not had the opportunity in any one case to give to it the prolonged study which it demands.

These cases are characterized by a special form of delusion which differs radically from the delusions either of grandeur or persecution. These latter incline rather to impel to action, the delusions of doubt restrain from it. The systematized delusion has something rather positive, aggressive, and self-assertive. The delusions of doubt are not so firmly fixed. They are changeable, negative, vulnerable, and confessed with timidity and mortification. They are somewhat allied to melancholic conditions, but are not characterized so much by depression of spirits (psychalgia) as by confusion and perplexity of mind, which exasperate the patient, and from which he would be only too glad to escape. He is apt to recognize perfectly the morbid character of the mental state, and will willingly take measures to counteract and cure it. Taxed with it he will even joke about it, and it might require a stretch of conscience to pronounce him a lunatic under the law. Hence it is a species of reasoning mania. Some cases hardly amount to delusions in the narrow sense; the patient does not so much believe a false thing, as he is impelled to solve innumerable problems which present themselves to his mind, and which may be as useless as they are perplexing both to himself and others, and equally imperative. They are exaggerated states of that intense impulse to solve a problem or discover a mystery which is not infrequently seen in a healthy mind, hence they are analogues, or perversions of healthy processes—as is the case always in insanity. Hence, again, they are *intellectual* in character (as is every true alienation) although this might be disputed by some in favor of an

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<sup>1</sup> Read before Am. Neurological Ass'n., Long Branch, July 21st, 1887.

amorphous moral insanity (so-called), because the follies and perplexities of these cases claim a superficial attention and are not sufficiently investigated and detailed. A surface view of such a case would not reveal its essential psychic features. This particular state (doubt, perplexity) unconfessed, probably lies at the root (forming the motive) of a certain number of eccentricities and aberrations which are ascribed by some to mere perversity of moral character, and by others are attempted to be shielded under the dark but ample cloak of moral imbecility. Other cases approach nearer the systematized delusion. They present certain morbid associations of ideas, which produce luxuriantly various crops of indecision. These patients link their fate to the trivial happenings of the hour; they will be lost if some unimportant and impertinent fact obtrudes itself suddenly upon their diseased imagination; they are somewhat like the gamester who stakes his fortunes upon the interval of time which it takes for a fly to traverse a pane of glass. The vacillation of their minds becomes amusing to the ignorant, but tragic to the immediate family who suffer; while it is both vexatious and exhausting to the patient, and highly instructive to his physician. All these aberrations of the so-called "intellectual sphere" shade into one another, and into others; in fact, they form an inextricable network of error just exactly as broad as the human mind. This condition, however, is not to be confounded with confusional insanity, for these patients have their wits about them: they are not incoherent, but quite connected in their ideas and speech, as indeed is necessary in order for them to accomplish the amount of hair-splitting and wool-gathering of which they are capable. There is, however, some likeness to the imperative conception, for these patients are impelled quite irresistibly to engage in their morbid penance or dialectic gymnastics, in order often to deliver themselves from the limbo of their own thoughts. They are bound to a rock Prometheus-like; as soon as their vitals are in, they are torn out again, or, if one spirit of doubt is laid a dozen arise to take its place.

Mrs. A. B., a resident of a distant city, was brought to Philadelphia, to be placed in a hospital for the insane. During the preceding year she had shown change of character and habits, and had developed some well-marked symptoms of the *Folie du Doute*. She has exhibited this in a variety of ways. She has been known to get in and out of bed at least twenty times on retiring at night because of some morbid train of associated ideas which made her feel that she had not performed the act aright, and that unless it was done in a certain precise manner it would in some way be the cause of disaster to her, producing disease or her death before morning. So again, recently she obliged her husband to remain in her room with her, after rising, until she had put on and taken off again, innumerable times, one of her skirts, apparently from a morbid impulse of the same kind. She has ceased reading the

newspapers and visiting her friends, because she has constantly presented to her mind, when reading or conversing, some idea which is immediately taken up, and serves as the starting point for another train of perplexing thoughts. In order to be rid of these morbid promptings she is impelled to resort to little tricks repetitions, and foolish actions, which explain the erratic conduct of which she is guilty. Thus, when dressing, she obliges her husband to hold a pin for her which she must seize at just the right moment, otherwise the toilet must be made again. She has sometimes insisted upon this long-suffering husband rising in the night, going down stairs, lighting a certain gas-burner and extinguishing it again in just a certain way, because this was necessary to absolve her in some manner from some of her morbid doubts and associations, and has not been content until her husband has humored her. On his return to bed he would be subjected to a long cross-examination on the subject before sleep could come to that troubled hymeneal couch. At night, she has seen printed sentences before her eyes suggesting disagreeable and depressing subjects—but these are not true hallucinations, as she does not believe in their reality. Yet they are a suspicious symptom, and probably rudimentary in character. When this patient reached Philadelphia, her husband drove her to a hotel, instead of to an asylum, and then told her of his intention to put her in restraint. The lady protested indignantly against this, stationed herself in her room as though to resist a siege, and bade her husband and the medical profession defiance. A few hours later I was requested to visit her. She was a young woman, about 30 years of age, rather under weight for her build, of not very good color and complexion, of sound muscular system, but not well developed, and suggesting rather physical than mental impairment, and that not serious. She had refused to have a physician summoned, saying to her husband that if he brought one she would not speak to him. Accordingly when approached she threw herself on a lounge and turned her face to the wall. This was evidently not in accord with her naturally frank and communicative disposition, and when assured that no wrong or injustice would be done to her, she at once sat up, shook hands and was henceforth herself. She conducted her share of a conversation on general subjects quite rationally, and soon branched off on suggestion into her eccentricities and notions. She said that she knew perfectly that her notions were morbid, and hoped to be able to conquer them if allowed to improve her health by horse-back and out-door exercise (a not irrational idea). She was bitterly opposed to going to an asylum : said it would fasten a stigma upon her for life : and even threatened to kill herself if taken by force (this was the first time she had threatened suicide). She said that she would oppose and resist physically being taken from the hotel to an asylum. The patient was very candid and courteous to the writer, but could not be persuaded to see it to her advantage to go voluntarily to the hos-



pital. She spoke of her own infirmities as calmly and intelligently as though they were the disorders of a third person. She confessed that they annoyed and distressed her, and presented problems to her mind which she could not solve, yet she knew these were morbid and foolish. It was the opinion of the writer after prolonged conversation with this lady, that it would not only be injurious to take her to an asylum against her will, but that it would probably create a public scandal. As the case was a most important one, and as the law in Pennsylvania requires two physicians to sign a certificate (if a certificate were needed in this case), I requested that Dr. C. K. Mills should see the patient in consultation. He was accordingly summoned, and after a most thorough examination, concurred in the writer's opinion, both as to the nature of the disease and the best course to pursue. A joint written opinion was given to the husband, which he could use in future, and he was advised to take his wife home for a further trial where, if in time the case grew worse, he would be more justified in resorting to legal measures. It is important to add that this lady had an insane first-cousin; and that her father had been an excessive drinker.

The several cases which I have seen, presenting these and similar symptoms, have all probably been of the neurotic or constitutional type. These things are not accidents, but develop in the soil of character and temperament. If we look within we may, perhaps, see occasionally the little germs whence grow these formidable thickets which rob the mind of its light in other people. It is perhaps not far from the vagaries of lunacy to the soul of the philosopher, for I believe it was said of Empedocles that he threw himself into *Ætna* in order to absolve himself from the doubts and mysterious associations that obscured his mind; but the volcano threw up his golden sandals and gave no other answer. I have known a true morbid condition of mind to exist in regard to the solution of mathematical, metaphysical, scientific, historic, and philological questions. One might suppose that they could be allowed to rest awhile, and even if never solved yet never be permitted to disturb the equilibrium of a mind which has other duties, interests, and pleasures. But some spirit of unrest seems to urge the mind into a mad race: many pull up in time and are safe, but some are not so fortunate and, like the wild herds of the plain, irresistibly pushed on from behind, fall over the cliff.

It has not been the intention in this paper to attempt the formation or advocacy of a new species of mental disease, but simply to describe certain symptom-groups, which are no doubt not isolated in any one case, but would probably be found associated with other manifestations of disease in any case which was followed through a long period of time.

## REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILADELPHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF F. X. DERCUM, M.D., VISITING  
PHYSICIAN, AND CHARLES K. MILLS, M.D., VISITING PHYSICIAN.

*Three Cases of Acute Mania Occurring in the Same Family (Folie  
Communiquée).*

Reported by Dr. Harriet Brooke, Assistant Physician.

These three cases have awakened considerable interest in Philadelphia, where they have been discussed in the newspapers. One of the three became depressed in mind and weak physically after child-birth or a miscarriage. A white woman, named Mrs. A—— L——, reputed to be a "medium" and "clairvoyant," was called in to try her powers on this patient. A neighbor told some story about her daughter having been very ill and having been cured by Mrs. L—— "voodooing" her. The medium practised her charms and incantations upon the patient, but instead of restoring her to health, caused her to pass into a condition of violent mania. The patient lived with her mother and a married sister. Mrs. L——, it is said, told the ignorant and credulous women of her miraculous powers, awing and terrifying them, and causing them to believe that an evil spell had been placed upon them. She gave them each a fetich or amulet, and instructed them to repeat certain phrases, such as: "Jack, drive all evil away!" She also administered a tea of some kind. Soon the three women were in a condition of violent acute mania, and all three were sent to the hospital the same day. The "medium" has since been arrested, and sent to prison, in default of bail, on the charge of violating an act prohibiting fortune-telling, etc. It was testified, at one of the hearings, that the medium gave something resembling a piece of wood to the first sick woman; that she produced a small muslin bag that she hung around the first patient's neck by means of a tape; that she told the mother that when the girl raved, she was to say to the bag: "Look up, Jack, be good to Eliza;" also that a battery was brought and electricity was administered to the head of the patient.

The following is a brief history of each of the cases from the records of the hospital:

CASE XVI.—E. M., aged 22 years, colored, married, born in Philadelphia, was admitted to the hospital August 12th, 1887. She has two children, both living and well.

The first symptoms of insanity were manifested three weeks before admission, when she became much depressed mentally. A medium was called in, as already stated, and in about a week she became maniacal and at times noisy and violent.

Examination and inquiry since admission show that she has lost much flesh since the beginning of the attack. Her pulse is weak. Her temperature was taken on the 13th, 14th, and 15th, with the following result :

Aug. 13th, evening (7 o'clock), 100.4° F.

Aug. 14th, morning (11 o'clock), 100°; evening (8 o'clock), 99°.

Aug. 15th, morning (11 o'clock), 101°.

The surface of the body has a tendency to become cool. Usually she is noisy, violent, and destructive; but becomes weak and exhausted, and will remain quiet for a time. She mutters incoherently; but little of what she says can be understood. Apparently she has hallucinations of sight and hearing. Occasionally, words can be caught, showing that her mind is running on the subject of witchcraft and the "white woman," as the medium is called by all the patients. Sometimes she can barely articulate, so great is her weakness and exhaustion.

CASE XVII.—R. D., aged 55 years, colored, widow, born in Delaware, admitted to the hospital August 12th, 1887. She has nine children living (two of them are the other cases of this series). She has been insane twice before at long intervals. She can neither read nor write. She is a devout member of the Baptist Church.

She had shown some peculiarities for several days, but the first active symptoms of insanity were manifested early in the morning of the day before admission. It is said that before the active outbreak, among other things, she went regularly to market and brought home a basket full of provisions which she would burn in the stove. Both of her daughters had evidently suffered from want of food.

On admission, she was exceedingly violent in actions and in manner—raving incoherently, destroying clothing, denuding herself entirely, striking at and struggling with her attendants, shouting and screaming. She constantly mistook the identity of the physicians, attendants, and persons who visited her. She sometimes seemed to imagine that she was conquering the devil by various incantations. Her physical condition was good, much better than that of her daughter (Case XVI.).

Temperature record for three days :

Aug. 13th, evening (7 o'clock), 99.8° F.

Aug. 14th, morning (11 o'clock), 101°; evening (8 o'clock), 99°

Aug. 15th, morning (11 o'clock), 98.6°

CASE XVIII.—S. D., aged 25 years, colored, married, born in

Philadelphia, was admitted to the hospital August 12th, 1887 (sister of Case XVI., and daughter of Case XVII.). It is stated of her, as of her mother and sister, that she has always been industrious and temperate. She has four living and two dead children.

She first manifested active symptoms of insanity the day before her admission, soon after her mother became maniacal. Her symptoms are and have been much the same as those of her mother. She is violent in speech and action, apparently has hallucinations of sight and hearing, raves and rambles incoherently.

Her mind is less completely clouded than in the case of the other two patients. She can be made with difficulty to talk a little about the "white woman" or "medium." She speaks sometimes of the "Jack" which the "white woman" put around Eliza's neck. She says it looked like a root. Most of the time, however, she talks and raves incoherently about matters that cannot be understood. Her physical condition is comparatively good.

The three patients have been put on conium, and carefully regulated nourishment.

## Society Reports.

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### AMERICAN NEUROLOGICAL ASSOCIATION.

*Nineteenth Annual Report.*

(Concluded from page 539.)

*First Day, Afternoon Session.*

The Association was called to order at 3:20 o'clock by the President.

On motion by Dr. E. C. Spitzka, of New York, the by-laws were suspended, and Dr. William A. Hammond, of New York, was elected honorary member, his name having been proposed by Dr. Charles K. Mills, of Philadelphia, and others.

DR. CHARLES L. DANA, of New York, then read a paper on

#### HEREDITARY TREMOR,

a disease heretofore undescribed, and consisting of a fine tremor constantly present during waking hours.

#### REMARKS ON DR. DANA'S PAPER.

DR. J. J. PUTNAM, of Boston.—I have seen these cases, one recently which conforms with what Dr. Dana says, and there is no other disease in the family. There is another example of this kind in our vicinity who has also tremor of the head that is not senile.

DR. F. X. DERCUM, of Philadelphia.—I have seen cases of this kind, and instances where the whole family had the same tremor. In one case there is also decided tremor of the head.

DR. GRAEME M. HAMMOND, of New York.—I would ask Dr. Dana if the form of tremor he describes is the only variety of tremor that he attributes to hereditary influence. I think he will recollect one of my patients at the Post-Graduate School and Hospital, a case of the choreic form of tremor which certainly

was hereditary. Dr. Seguin also saw the case. It was impossible for the man to sit upon a chair, he was unable to walk, and the whole case was very similar to chorea in its manifestations. The patient could only trace the disease as far back as his mother, who had it and died in consequence of it. A brother had the same disease and also a sister; and a niece whose father did not have it. After a certain length of time they all died, as far as could be ascertained, from the disease. It did not appear in this man until about the age of thirty-five years, and then progressed rapidly up to the state in which I saw him and there remained. He had also mental failure towards the end, delusions of various kinds, etc., and the other members of his family had been afflicted in the same manner.

DR. DANA.—I recollect Dr. Hammond's case, but I was not aware that it had an hereditary character. I regarded it as *tic convulsif* rather than tremor. At least I did not regard it as belonging to the type which I have described, and which I think is rare. A good many persons have considerable neurasthenic tremor for a considerable portion of their lives, which is not hereditary. The peculiarity in this class of cases is their very striking hereditary history. In some cases which I saw, the head under conditions of great excitement or extraordinary nervous depression oscillated, but not ordinarily.

DR. GEORGE W. JACOBY, of New York, read a paper on

MICROSCOPICAL STUDIES IN A CASE OF PSEUDO-HYPERTROPHIC PARALYSIS.<sup>1</sup>

REMARKS ON DR. JACOBY'S PAPER.

DR. F. X. DERCUM, of Philadelphia.—I have been very much interested in the theory and the pathological report made by Dr. Jacoby. I had occasion last autumn to examine a case of atypical muscular hypertrophy, occurring in an adult, which showed irregular places of hypertrophy, but not pseudo-hypertrophy. I also saw a case in which there was some hypertrophy of the connective-tissue elements and changes in the muscles. I have seen cases of interstitial myositis and myositis proper, but whether the connective-tissue changes are secondary is difficult to say. The drawings exhibited by Dr. Jacoby, however, answer the question in his case almost beyond doubt. In the specimens which I

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<sup>1</sup> See p. 577 of this volume.

studied I used osmic acid to determine whether or not there was fat in the muscle fibres, and the droplets were present in rather large percentage.

DR. J. J. PUTNAM, of Boston.—One remark which Dr. Jacoby made calls to mind an interesting point which might be further commented on, and that is a mixture of the degenerative process and the true inflammatory process. Certainly it seems to be marked here, and it was in a case of hereditary ataxia in which I examined the spinal cord. Besides the marked changes, unquestionably due to abnormal development of certain portions of the spinal cord, there was evidence of true inflammatory action. Also we see something similar in the degeneration of nerve fibres with changes in the connective tissue surrounding them.

DR. B. SACHS, of New York, then read a paper

“ON ARRESTED CEREBRAL DEVELOPMENT, WITH SPECIAL REFERENCE TO ITS CORTICAL PATHOLOGY.”<sup>1</sup>

REMARKS ON DR. SACHS' PAPER.

DR. C. K. MILLS, of Philadelphia.—A paper of this kind should be discussed, and yet it is difficult to pick out the points for discussion. The observations are very interesting and important, especially the histological or pathological portion. The facts which Dr. Sachs has so clearly demonstrated prove that, in these cases, the changes are primarily the result of inhibition of the development of the nervous elements proper, and not the result of some preceding inflammatory process. There may be observations of this kind upon record, but they are few, if any, and added to the work which has been done in this society and elsewhere on the gross condition, it marks an important advance in the study of the pathology of idiocy. I congratulate Dr. Sachs on the manner in which he has presented the case for our consideration. We wish to go still further and eventually have studies not only of the fissuration of the cerebral surface and its histology, but of the histology of the entire cerebrum and spinal tracts. I should be glad to hear from Dr. Dercum, who has been doing some work in this direction.

DR. F. X. DERCUM, of Philadelphia.—I have not done much work on idiots' brains, but something in the study of the brains of the insane. The only brain of an idiot which I have examined

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<sup>1</sup> See p. 541 of this volume.

was not one of inhibition of development, because there was marked sclerosis of the skull and enormous thickening with external ossific pachymeningitis, and also the pia mater was very much adherent, these changes doubtless being secondary to the inflammatory condition. What I saw microscopically were distorted cells, with processes atrophied, nuclei absent, and I found also a certain amount of proliferation of the neuroglia.

DR. MILLS.—The report of this case will help us a little further on, possibly, in the classification of idiocy. The classifications which we have in ordinary text-books are of no earthly account for scientific purposes. If we go into any of our large Asylums for feeble-minded children and attempt to study the cases from the stand-point of our present classifications, it is almost, if not entirely, impossible to do so. Studies of the kind Dr. Sachs has made will help us in that direction, and if continued we may soon arrange a respectable classification for idiocy, instead of having to consult such old-timed classifications as those of Ireland and others.

DR. R. W. AMIDON, of New York.—If I understand him rightly, Dr. Sachs argues that the absence of any gross lesion on autopsy is against there ever having been gross lesions present.

That may be perfectly true regarding inflammatory processes or cerebral lacerations, etc., but I do not think that it can be true with respect to hemorrhages, especially intra-meningeal hemorrhages. I have seen two cases which were very much like this, and in which there was found on autopsy no gross lesion apparent, but in which there was almost indubitable clinical evidence of previous existing hemorrhage. In my opinion, hemorrhage of considerable size, and in such location as to very much impair the growth of the central nervous system, may occur and be so completely absorbed as to leave no trace that could be seen at autopsy. While I have no distinct incontrovertible proof that such is the case, that is my impression, and I would like to ask Dr. Sachs if that has occurred to him as a possible condition that existed and inhibited the growth of the centres, and left a change which at the time of the autopsy seemed to be developmental.

DR. SACHS.—I had thought of an intra-uterine causation, but as to meningeal hemorrhage as the cause, I do not favor that view for the reason that an intra-meningeal hemorrhage sufficient to cause these widespread changes would have had to cover almost



the entire cortex, for these histological changes were noted in all parts of the cortex, and there would have been other and more serious changes following in the wake of such an hemorrhage: we might have expected thickening of the meninges, changes in the blood-vessels, etc. Besides, the clinical condition would have been reversed, and the child would not have been brighter at birth than two or three months afterwards. If it had been due to an intra-uterine hemorrhage, the mental condition would have been very poor at birth and improved somewhat afterwards. I have no doubt that the traumatic factor had something to do with this inhibited development, possibly through changes in cerebral circulation, but how this was effected is still a mystery. There was no convulsive movement, which is an exceedingly important factor in computing the presence or absence of cortical irritation in the meningeal thickening. It seems to me that there was a process away from the meninges.

I would like to say that during life I had concluded that this process was not meningeal, and could not be in the ordinary line of idiocies. In this case I argued that the process was in the cortex and not an irritative lesion upon the surface. My idea is that the cells developed to a certain stage and that from that time on, whatever the cause may have been, their growth was inhibited. For that reason we find but few cells which show atrophy, *i. e.*, cells which had grown to their full extent and then had undergone retrograde changes.

DR. DERCUM.—The paucity of the blood-vessels as compared with the number present in normal sections is very apparent.

DR. SACHS.—I have found a fair number, possibly the ordinary number, of perfectly normal capillary vessels in all the sections examined.

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*Thursday, Second Day, Morning Session.*

The Association was called to order at 10:30 o'clock by the President.

DR. JAMES J. PUTNAM, of Boston, presented microscopic sections with a report of a case of

**"SARCOMA INVOLVING THE INTRAPELVIC NERVES."**

The following case is offered as a contribution to the clinical and pathological history of "paraplegia dolorosa," due to the invasion of the intra-abdominal nerves.

The facts observed harmonize fully with those reported in the recent excellent summary and analysis by Dr. R. W. Amidon,<sup>1</sup> but the case is worthy of record because of the very late appearance of anything that could be called cachexia or of the enlargement of the lymphatic glands; because of the secondary and indirect involvement of the spinal cord; and because of the very notable fluctuation of some of the symptoms.

The case is that of a gentleman of seventy-two,<sup>2</sup> of excellent previous history and habits, and no family tendency which could be expected to make him liable to sarcomatous disease.

The first symptoms showed themselves about one year and a half before his death, and consisted in pain in the middle toe of the left foot, recurring frequently for some months, and occasionally throughout the remainder of his life.

Six months later, the whole left leg and the sacral region became severely painful, the pain confining itself, however, to the posterior and outer surfaces of the thigh, the outer side of the leg, and the outer half of the foot. The pain was accompanied with some paræsthesia, and this was especially true of the outer half of the sole of the foot, which felt, when he trod, as if there were small marbles beneath the foot, a sensation which never wholly left him.

Although a bilateral distribution of the symptoms is usually so early and so valuable a guide to the diagnosis, the right leg was not attacked in this case until nearly six months after the left.

Meantime, the pain in the left leg had greatly lessened during a trip to Florida.

I first saw the patient six months before his death, and six weeks previous to my visit pain, constant and of a grinding, aching character, had begun in the right leg, confined mainly to the posterior and outer sides of the thigh, and had renewed itself in the left leg.

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<sup>1</sup> "Malignant Disease of the Spine," etc., N. Y. Med. Journ., Feb. 26th, 1887.

<sup>2</sup> Seen by me in conjunction with Dr. Norton Folsom, of Cambridge.

Walking had become difficult on account of progressive muscular weakness; this weakness was found to affect not only the muscles supplied by the sciatics, but also the extensor quad. cruris, and the psoas and iliacus. The muscles of the calves were relatively free. All the affected muscles were somewhat atrophied, but none of them had lost their faradic reaction or showed any degenerative response to galvanism. A very slight diminution of tactile sensibility was found either at this examination or somewhat later, in the skin of the buttocks, in the vicinity of the arms, but nowhere else.

Cutaneous hyperæsthesia was not present to any marked degree, and yet he found applications of galvanism, even of moderate currents, so very painful as to suggest an unnatural sensitiveness in this respect.

This absence of marked anæsthesia, combined with the fact that there was but little sensitiveness to deep pressure in the affected muscles (the thighs only were somewhat sensitive), and none along the nerve-trunks, in spite of the great pain and weakness, was significant as against a primary multiple neuritis, even had the distribution of the pain favored that diagnosis.

No cachexia or anæmia could be detected, and none showed itself at any time until within two or three months of the patient's death, when he was exhausted by pain and discomfort. Indeed, it is to be remembered that a purely local sarcoma, as this proved to be, not involving the organs of digestion, need not seriously impair the nutrition or the constitution of the blood for a long period.

The knee-jerk was absent on both sides.

No tumors could be discovered by rectal examination.

There was no disorder of micturition either at this time or later.

Several examinations of the urine were made with negative result, except that lead was found on two occasions and by different chemists.

This examination for lead was made at the suggestion of Dr. S. G. Webber, who saw the case in consultation with Drs. Folsom and Wyman and myself as a *possible*

cause of the neuritis. The entire absence, however, of symptoms referable to lead-poisoning makes this an interesting case, in evidence of the fact that lead need cause no symptoms, even when present, as one of these analyses showed, so as to be eliminated in considerable quantity.

During the seven months, from my first visit until the patient's death, the disease made on the whole steady progress; but it is noteworthy that the left leg, which was the first to be attacked, became after a time almost wholly free from pain, and remained so for several months, though it did not recover its strength. It was also noticeable that no glandular enlargements could be detected until about two months before death, when a bunch was noticed in the right groin which rapidly increased in size. Finally the new growth made its appearance in the skin over the lower part of the back, the buttocks, and eventually the abdominal walls. Before the actual discovery of the growth, the elements in the diagnosis were signs of neuritis involving a number of adjacent nerves on both sides of the body, causing progressive weakness with severe but fluctuating pain, yet without complete paralysis, and the absence of evidence of acute generalized idiopathic neuritis, both in respect to the distribution of the symptoms, the rapidity of the muscular atrophy, or the electrical reactions.

Autopsy by Dr. H. Fitz.

Dense nodulated tumor in right groin (infiltration of skin of back and abdomen not mentioned).

Head not opened.

Nothing abnormal found on examination of heart and lungs, excepting œdema of latter and pleural adhesions.

Old peritoneal adhesions about liver; no calculi. Liver fatty infiltrated.

Spleen hyperplastic; normal size.

Kidneys somewhat atrophied.

Bladder dilated; contains half a pint of urine; prostate moderately enlarged.

Nothing abnormal in stomach and intestine, with the exception of a sarcomatous infiltration of a loop of ileum

some three inches long; no special constriction of the calibre; slight ulceration of the surface.

The perinephritic fat tissue, the lumbar muscles, the pelvic connective tissue (moderately), the muscles and glands in the inguinal and iliac regions infiltrated with a homogeneous grayish-white dense tissue. The sciatic and lumbar nerves, especially the right, as they emerged from the pelvis are infiltrated with the same tissue.

The vertebræ were not eroded. The lumbar portion of the spinal cord was removed for examination; on inspection it appeared healthy. The structure of the growth was largely cellular; the cells round, somewhat larger than leucocytes, and contained single and double nuclei; the intervening fibrous tissue moderate in quantity. The structure was regarded as that of a sarcoma, round-celled.

The clinical points of chief interest relate to the early diagnosis of this serious disease, and in this respect the signs of severe and yet partial irritative neuritis seem to me eminently important, and later the fluctuating character of the symptoms. One pathological point concerns the relation, which I think is a close one, between the neuritis and poliomyelitis—a relation which shows itself in a number of ways.

#### REMARKS ON DR. PUTNAM'S PAPER.

DR. C. K. MILLS, of Philadelphia.—I did not hear the fore part of Dr. Putnam's paper, but as I understood it the case was supposed at one time to be one of sciatica. The report is interesting in several particulars. First, with reference to the question of diagnosis. Several years ago, my attention was directed to the fact that the diagnosis of sciatica was occasionally made when there was intra-pelvic sarcoma, simple or involving the bones of the pelvis.

One of my lectures at the Blockley Hospital in Philadelphia was reported in which I took occasion to give the history of a case of supposed sciatica, but which proved to be one of intra-pelvic sarcoma. I have seen several of these cases; one at St. Mary's Hospital with Dr. M. O'Hara, where the same diagnosis of sciatica had been made, but the continued examination showed

that it was undoubtedly a pelvic sarcoma. In the Philadelphia Hospital, also, a patient had been treated in different departments, and in all it was supposed that it was a case of sciatica. Dr. J. W. White and myself finally made a careful examination, and we found diffused disease of the acetabulum, and at the autopsy it was demonstrated that it was malignant in character.

The differential diagnosis of sciatica is one of practical importance, and the suggestions with reference to neuritis and poliomyelitis I regard as of considerable interest. My intention had been to bring up that part of the subject for discussion at a later period in the meeting.

DR. F. T. MILES, of Baltimore.—In a case like this it is well to get all the information we can, so I will briefly outline the history of a case of malignant disease of the pelvis. A member of Congress fell twice in the streets of Washington, striking upon the same hip, and received contusions which were followed by intense pain. After suffering from severe pain for some time, the case was called one of sciatica, and he was brought to Baltimore to see me. I then looked for symptoms of neuritis, but there was no wasting of the muscles or weakening, and the response to the faradic current was very good—there was no reaction of degeneration. There was nothing abnormal about the pelvis that could be detected, and I examined him not only externally but per rectum, for I think that such intense pain would naturally suggest the possibility, at least, of the presence of a malignant growth upon the nerve which gives rise to the severe pain. Nothing whatever could be detected. As the patient had, in addition to his severe pain, and superinduced by it, a love for morphine, the question was as to how far his statements could be taken as actually true concerning his sufferings. As there was no evidence, either by pressure or otherwise, that there was neuritis, I reached the conclusion that it was a case of malignant growth or the expression of pain which did not exist, but made for the purpose of securing the usual quantity of morphine. I inclined to the opinion, however, that it was a case of malignant disease, as it had none of the usual symptoms of neuritis. The point of interest is that in general neuritic trouble we find with loss of power atrophy of the muscles, and in the second case there was but little atrophy although the nerve was involved. My patient went home, and died afterwards with the development of a malignant tumor about the hip. A competent surgeon living in his locality sent me word

that there was obviously a growth of malignant disease involving the hip. The sole symptom when I saw him was the intense pain, for the relief of which morphine was required.

DR. PUTNAM.—Was there any evidence of cachexia in the case?

DR. MILES.—Cachexia was absent.

DR. MILLS.—In one of my cases it was present, and in the other two it was absent.

DR. JAMES HENDRIE LLOYD, of Philadelphia, read a paper

“ON A CASE OF INSANITY OF DOUBT”<sup>1</sup>

REMARKS ON DR. LLOYD'S PAPER.

The President, DR. L. C. GRAY, of Brooklyn.—With regard to this case, I have no doubt as to its being one of insanity, and I should not have the slightest hesitation to commit such a person to a lunatic asylum. But I would not do it unless I knew something about the case. They are extremely dangerous, in my experience, because a number of these patients will go for a long time and present no tangible symptoms which will warrant condemning them, and some morning they will do some horrible deed which shows that they have been all this time just on the ragged edge of insanity. I recollect one case particularly, that of a woman who was brought to me who had been in this condition for several years. At one time she washed her hands four hundred times in one day by actual count and yet was sane, but nevertheless, after she had been run down in general health and had suffered from neuralgias which were somewhat benefited by treatment as was also her mental condition, I made up my mind that the best thing to do was to send her to Bloomingdale Asylum. I explained to her that she was to go there and why she was to go there and in all this she agreed with me. But the day before she went she got into a dispute with her mother about some little thing, lost her temper, and as soon as her mother left the room she took a piece of a match from under one of her finger nails, got a piece of paper, lit the match, set fire to the paper and then to her clothing, and burnt herself in a most terrible manner, thus showing that she had been secreting under her finger nail the end of a match and a bit of paper ready for any moment she might have opportunity to use them. That illustrates the danger in these

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<sup>1</sup> See p. 590 of this volume.

cases, so that in any case where there was the slightest doubt of the ability of the friends to take charge of them, the danger of their being left at home would make me unhesitatingly commit them to an asylum, and take the risk which we all have to take in the management of insane cases.

DR. LLOYD.—I do not wish to be misunderstood as to my position regarding these patients. They are pre-eminently proper cases for restraint, such as an asylum affords if you can get them there. This woman refused to go, and could not be removed from the hotel without great resistance and being carried through the most populous part of Philadelphia. They came to our city and stopped at the Continental Hotel, and it was there that the husband told her what he was going to do with her. If by any management we could have gotten her to the asylum without raising a tumult I should have had her removed. But she was present and recognized her own condition, had perfect control of herself, and would have demonstrated to a lunacy commission that she was irregularly detained. I think that we have a right to regard the policy as well as the absolutely scientific requirements in some of these cases. The so-called intermediate treatment would probably have been best for this patient. That is, there are certain forms of house retreats where patients can be taken without being declared lunatics under the law.

The PRESIDENT then read a paper on

“CHOREA”

in which he maintained that it is not the trivial disease it is generally supposed to be.

REMARKS ON DR. GRAY'S PAPER.

DR. M. ALLEN STARR, of New York.—I would like to ask Dr. Gray if he has any facts which bear on the statement made by Charcot with regard to the danger of adult patients suffering from chorea going into a condition of insanity, especially melancholia. I have had under my care four cases of chorea in adults over the age of thirty years, and in one of these cases there seems to be a marked tendency to melancholia. According to the statement made by Charcot, that seems to be the tendency in a large number of cases of chorea occurring in adults.

DR. JAMES HENDRIE LLOYD, of Philadelphia.—I had a severe



case of chorea occurring in a pregnant woman a few years ago, so severe that it was impossible for her to lie in bed, and there was a decided degree of mental impairment, which I described as mild imbecility—she was so foolish and silly. There was a suspicion of syphilis in the case, and her general condition was poor. She improved decidedly under the influence of the *four chlorides*, of arsenic, iron, bichloride of mercury, and hydrochloric acid. She eventually got vastly better, but did not make a complete recovery.

DR. F. X. DERCUM, of Philadelphia.—I would like to ask Dr. Gray what his experience has been in the use of cimicifuga. In the University Hospital we are in the habit of giving our choreic patients arsenical preparations or combinations of arsenic and iron, but occasionally we meet with an intractable case, when we fall back on cimicifuga, and not infrequently with good results. It has appeared to me that the cimicifuga was the most efficacious in girls about the age of puberty.

I would also ask Dr. Gray whether he has had cases of chorea associated with epilepsy. A case was brought to me of a child suffering from epilepsy which was brought rather rapidly under control with bromides, but after a time general and marked chorea developed. Whether this was due to the depressing effect of the bromides or to other causes I am unable to say.

DR. GEORGE W. JACOBY, of New York.—I have under treatment a woman 38 or 40 years of age who is affected by stuporous insanity, the primary curable dementia of the Germans, and she has been affected in this manner eight weeks. Preceding the mental symptoms, she had an attack of chorea develop in consequence of sudden fright, and she has at present choreic symptoms in addition to the symptoms purely of stuporous insanity.

I was struck by one remark made by Dr. Gray when speaking of treatment, and that was with regard to rest. Almost all writers on chorea seem to advocate different kinds of treatment, and some in distinct contradistinction to what Dr. Gray has advanced. For example, French writers advocate massage and active movements, and endeavor to teach patients to co-ordinate. I have not had any personal experience in this direction, but it struck me as being rather interesting that two directly opposed plans of treatment are equally recommended.

DR. F. T. MILES, of Baltimore.—I will add a case of insanity and chorea combined in a puerperal woman. She was dangerously

insane, and once tried to throw herself out of the window, and once tried to swallow some medicine which she supposed was laudanum. She died without material improvement in her condition.

With regard to rest, one of the worst cases I ever had was that of a boy sent from a public school when the physician had tried the treatment by faradism thoroughly every day, besides walking the patient up and down between two attendants. Under this treatment the boy got a great deal worse. My own opinion about rest is that it is an important remedy. It is not only rest of body, but rest of mind, and that even the introduction of playmates to give a pleasurable excitement is bad.

DR. GRAEME M. HAMMOND, of New York.—With regard to the relation between insanity and chorea, there may be a distinction as to whether the chorea follows the insanity or the insanity follows the chorea. Charcot's cases were those in which insanity followed chorea.

I referred yesterday to a typical case of what Charcot has described as hereditary locomotor ataxia, the disease being present in the mother, in a brother, sister, and himself, and in a brother's child. It did not appear in any of the cases until the patients were adults, and my patient was forty-seven years of age and had had the disease one year. He told me that other members of the family had had chorea and died insane, and while I had this man under observation he became insane and remained in that condition, and when I saw him last he showed evidence of violent insanity.

With regard to the treatment of chorea, it is a disputed point as to the beneficial effect produced by arsenic. Some use no other remedy; some never use it. I use it almost exclusively, and it would seem to be a remarkable coincidence that the large number of cases treated with arsenic should happen to get well while the arsenic is being administered. Rest is also efficacious. I also use iron and cod-liver oil, and these with arsenic and rest constitute a more efficacious plan of treatment than any I have ever tried.

DR. WHARTON SINKLER, of Philadelphia.—I think that in almost all cases of chorea there is more or less mental impairment. The patients are irritable, they are easily annoyed, and frequently girls are hysterical. Some cases will get well with or without treatment. But in the cases which have continued for several

months, the ability of self-cure has passed, and if they improve under any particular drug we should feel satisfied that that drug has benefited them. In dispensary patients, the opportunity is not afforded of giving attention to rest and hygiene, which are of benefit, but we have to depend almost entirely upon drugs. The drug which has been more constantly beneficial than any other with me is arsenic, and I have given it in large doses sufficient to produce its toxic effects, but have not seen any permanent ill effects in any of the large number of cases I have treated.

We have to bear in mind the fact that there must be organic changes in different parts of the system, and Dr. Osler has been making some very interesting observations, about to be published, with regard to the condition of the heart in chorea. He has examined a large number of patients at the infirmary for nervous diseases, who have remained free from chorea for several years, and has found in a large proportion some organic disease of the heart as a sequel. From these observations the cardiac murmur so frequently met with in chorea should not be regarded as simply a functional or hæmic murmur.

DR. R. T. EDES, of Washington.—I can recall two cases of severe chorea, in one of which the patient died; another case in which the patient passed into a condition of stupor that lasted for several weeks with final recovery; and finally another patient who, I think, would have died without treatment. In this case, the treatment was largely chloral to produce sleep, and conium to control the violent movements, and I think that these drugs had a great deal to do with the patient's recovery.

DR. GRAY.—With regard to Dr. Starr's question as to the association of chorea and insanity in adults, according to my observation choreic insanity is a rare condition. It is a form of insanity which I think many alienists have not seen much of. The cases of choreic insanity which I have seen have been in adults and the chorea has been simultaneous with the outbreak of the insanity. The cases which I have seen have also been the peculiarly chronic stupid passive forms of insanity that are usually harmless, which last for many years, until the family and the physician have given up all hope when the patient gradually recovers. As to whether an adult having chorea for the first time is more predisposed to insanity, I am unable to say. There is a strong distinction, however, between true choreic insanity and the apparent imbecility of the choreic patient. The brightest of choreic

patients may look like insane persons; but, as a rule, I do not think that they are impaired in their mentality. That form of something that looks like imbecility, which is impressed upon the choreic patient, is entirely different from choreic insanity in which the muscular movements are very slight and fibrillary, and very apt to be overlooked. Then, too, there is a difference to be noticed with regard to races. The grave forms of hystero-epilepsy described by Charcot have not been seen in this country, and it is possible that there may be a difference between our American people and the pure Latin race which makes up the French people.

That cimicifuga is one of the tonics which has a beneficial effect in chorea I have no doubt, but I have not been able to say more than that concerning it.

The association of chorea and epilepsy has often been described.

As to the question raised by Dr. Jacoby and the plan of treatment recommended by Germain Sée, I think he should raise the question, Why does Dr. Sée differ with me? There is no question, however, in my own mind as to the efficacy of rest. I have seen it over and over again. I am so fully convinced of its efficacy that I will not consent to take charge of patients unless they will agree to submit to the rest treatment. But you may do what you choose, these children will toss about their beds, get out of bed and run around the room as soon as the back of the attendant is turned, etc.; so it is only moderate rest that you get, and nothing like the rest secured in an adult patient. But as to the gymnastics, and muscular exercise, I have tried them and the results have been so indubitably deleterious that I believed I had no right to jeopardize the safety of the patient by persisting in them.

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*Thursday, Second day, Afternoon Session.*

The Association was called to order by the President.

DR. FRANCIS X. DERCUM, of Philadelphia, read a paper entitled  
TWO CASES OF HEMICHOREA ASSOCIATED WITH BRIGHT'S DISEASE.<sup>1</sup>

REMARKS ON DR. DERCUM'S PAPER.

DR. R. T. EDES, of Washington.—I should agree with Dr. Dercum inasmuch as I should not be inclined to regard the association of these symptoms with Bright's disease as altogether acci-

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<sup>1</sup> See JOURNAL OF NERVOUS AND MENTAL DISEASE, vol. xii., p. 473.

dental, but I should not explain them as he does, that is, as a kind of uræmic symptom, and in my belief, it is a mistake, frequently made, to ascribe so many of these nervous phenomena to uræmia. They have nothing to do with the accumulation of whatever it may be that gives rise to what is known as uræmia. It seems to me that these cases, and so with a great many cases of so-called chronic uræmia, are caused by a general disease and not by inflammation of the kidneys; that the entire arterial system is involved. In both of these cases there was distinct weakness on the side of the chorea, and I should think there was a lesion in the brain, or some kind of temporary interruption of circulation. We know that there are cases of hemiplegia which come and go rapidly, and in which we would not find anything obvious to the naked eye, should we have opportunity to examine the brain. In general paralysis, there is a hemiplegia which is spoken of as due to congestive attacks, but whether congestive or not, they probably have something to do with disturbances of the circulation. I should be inclined to explain Dr. Dercum's cases in this way; that these are cases of hemichorea occurring in Bright's disease, as we frequently have in Bright's disease hemiplegia due to the general arterial disease upon which the disease of the kidneys depends; that is, we have chorea instead of hemiplegia.

DR. C. K. MILLS, of Philadelphia.—I am strongly inclined to agree with Dr. Edes with reference to these cases, and not to agree with Dr. Dercum's view, namely, that in some way not clearly understood, Bright's disease acts so as to produce a unilateral impression upon the general nervous system and thus lead to these abnormal manifestations. At the Philadelphia hospital, I have seen many cases of Bright's disease and uræmia, and not a few cases of hemiplegia with Bright's disease, and in some of these have had opportunity to make autopsies. The explanation, it seems to me, is most likely a mechanical one, springing out of the conditions which are present within the cranium, in cases of uræmic apoplexy. These uræmic cases are cases of more or less general cerebral œdema. What is the result when there is general cerebral œdema? Certainly you have, in many cases, the œdema causing effusion into the inner cerebral membranes. It is common at post-mortem examinations to have this patent evidence of œdema in this peculiar appearance, disappearing as the effused fluid leaks from its peculiar positions. It seems to me that it is impossible, almost impossible to have such an œdema

without certain local effects. We have hemiplegia produced in the way which Dr. Edes has indicated; that is, in some parts of the brain there are mechanical effects produced by more marked effusion. The pia mater itself is practically a mesh of blood-vessels of considerable magnitude, and minute arterial hemorrhages occur which are easily overlooked; and there may be greater pressure from œdema at one part than another. In other words, there is absolutely an affection of the circulation or an interference with it in some way. Why it selects the left or the right side of the cerebrum is of no account here. Hemichorea, it seems to me, can be produced in this manner.

DR. DERCUM.—Although the view expressed by Dr. Edes is plausible, the fact that I discovered neither gross nor microscopical lesions, and the fact also that the French observers have not found lesions beyond œdema, makes me hesitate with regard to ascribing the symptoms to lesions where none is apparently present. Minute hemorrhages could not have escaped notice in my examination. Raymond's experiments on dogs certainly show a general impression that acted upon the nervous system the two halves of which have unequal powers of resistance. It is probable that, in one of the cases which I have reported, the patient may have, by this time, chorea on the opposite side. The œdema was equally well marked throughout the brain I examined.

DR. MILLS.—I would like to ask Dr. Dercum if he has noticed whether or not convulsions or paresis are more likely to occur upon one side than the other.

DR. DERCUM.—In my cases, one was affected upon the right side and the other upon the left. I believe that there was no special difference noticed with regard to sides by the French observers.

DR. E. C. SPITZKA, of New York, read a paper on

#### ACUTE OR GRAVE DELIRIUM.

It is unnecessary for me to define before this Association what is meant by acute or grave delirium: a condition which, while it shares many of the features of delirium due to febrile toxic agencies, is yet known to be of independent origin.

To some extent its peculiarly fatal character, and the essentially cerebral location of the lesion to which it is due, was recognized by Abercrombie, and particularly by

Thomas Mills who wrote in 1816. Speaking of a patient who, in consequence of domestic affliction and disappointment, developed a so-called brain fever, dying on the twenty-first day of the same, he says: "By some the disease would be denominated typhus, by others, from the appearance of petechiæ and the violence of the symptoms, the spotted, putrid or malignant fever." On dissection, no evidence of putrescent fever was found in the body; the thoracic and abdominal viscera were sound; but the brain exhibited "marks of excitement and inflammatory action. . . . The pia mater was highly vascular, and there was, moreover, on the surface of the brain a considerable degree of venous turgescence." One of the first to recognize it as a form of insanity distinct from mania was Luther Bell, of the Massachusetts Asylum, who termed it typhomania. Since his time it has been repeatedly described. The French and German alienists of the last decade were inclined to regard it as a clinical entity. Fürstner, Mendel, and Jolly consider it merely as a symptom which may be due to different pathological states. There is no question that some cases of acute fatal hysteria approach it so closely as to be clinically undistinguishable from it; while on the other hand, it may be a complicating culmination of ordinary insanity. It may also be an evidence of ulcerative endocarditis, of microparasitic invasion of the brain (as in one of my cases), and finally it may be entirely and exclusively the result of mental worry, domestic affliction, nay, of fear and expectant attention. There is no doubt in my mind, and Dulles verbally expressed the same opinion to me, that some of the cases classed as "hydrophobia" and rabies were cases of acute delirium, due either to the latter cause alone, or tinged with the prevailing newspaper epidemic.

As I have recently read a paper dealing with the etiological and clinical features of this disease elsewhere,<sup>1</sup> I will content myself with giving a rapid sketch of the progress of typical cases.

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<sup>1</sup> American Medical Association, June, 1887, at Chicago.

Ordinarily the disease begins with insomnia, malaise, and inability to think, accompanied by a bursting sense of pressure in the head. Increasing irritability and a sense of impending misfortune precede the outbreak, which latter is often so sudden as to suggest the fulminating type of typhus or of epidemic meningitis. In such cases, the patient, arising from a confused sleep, staggers round as if drunk; or having lain down oppressed by some real grief or subjective melancholy, he or she rises hilarious, dances round, vociferates, indulges in erotic or expansive imagery, to pass into apathy either with or without the intervention of a lucid remission. Soon the flight of ideas increases: in some, a wild aggressive delirium, marked by celestial, diabolical, or flaming visions, auditory hallucinations of voices, of thunder, and illusions of touch, such as that the skin is covered with vermin, preponderates. In others, there is a distressing anxiety from the onset, which, as it deepens, becomes a panphobic delirium; police officers, murderers, toads, goats, negroes, fleshless skeleton hands crowd round the patient, and shrieking, he or she jumps out of the window, beats the plaster from the wall and eats the bedding, and clutches the attendants with the frenzy of despair. The more violent class may sing, whistle, yell, and tear off their clothing continuously for days. Those who early suffer from impaired consciousness exhibit a suffused, stupid face, and lie almost motionless, groaning, or puffing and blowing with their mouths in a peculiar manner. Imperative movements, particularly of the variety known as *manus ad genitalia* in meningitis, but of far greater intensity, are next developed. In some patients, they take the form of salaam movements. In others, the head is kept plunging away at the ceiling till it is beaten to jelly. One case observed by me in a pauper asylum, rubbed his thumb knuckle against his teeth till the member hung by a thread of skin. In another group, an enormous reflex excitability is developed, and to some extent, and at same time, this is observed in all cases. The slightest touch suffices to produce an extreme flexion contracture in some, and as intense an opisthoto-



nus in other cases. In most, this reflex excitability extends to the pharynx, constituting an insuperable obstacle to forced alimentation. The few cases that have recovered with life, were such in which nutrition could be kept up, and in the history of the only case I am acquainted with where a complete cure was effected (one reported in Dr. Mills' service at the Pennsylvania Hospital by Dr. Harriet Brooke), special attention is directed to the readiness with which the patient took food as an explanation of the recovery, and as an exceptional feature.

The hurricane overwhelming the mental functions in grave delirium does not leave the somatic functions untouched. The temperature rises to from  $102^{\circ}$  to  $105^{\circ}$ , an acceleration of the pulse up to 140 beats accompanies this rise, and its character is thready, compressible, and it is sometimes irregularly intermittent. Peculiar trophic lesions, pemphigus, bullæ, pustules, and bed-sores complicate the picture, and during the last days or week of life a marked cyanosis is noted. The suggillations, occurring as a result of the injuries self-inflicted in the course of the patient's incessant rolling about, exhibit a resemblance to the hypostatic patches of the dead rather than the bruises on a living body. Everything indicates a profound exhaustion of the somatic forces, and death finally ends the history, either suddenly in the midst of the patient's screams, in collapse during stupor, or finally with every indication of oblongata paralysis during a lucid interval.

These lucid or paralucid intervals are the most remarkable features of the disease. They are noted in the initial period, during the development and progress of the delirium, and even at its end. No more pathetic picture can be conceived than the return to lucidity, just before death, of the mother of a family conscious that she is about to die, taking leave of her children, and distributing presents to the servants to atone for her delirious violence to them. This particularly when, as in one of my cases, the disease was entirely due to emotional causes, namely, the sudden death of the husband. Less perfect remissions are common during the very height of the disorder. The patient

awakes from a doze, recognizes that he is in a hospital or in an asylum, admits that his head is confused, or that he has been or is deranged, and, intermingled with these admissions, rambles about dogs, soldiers, snakes, and other objects. The inexperienced have on more than one occasion been induced to suspect simulation from this apparent inconsistency.

The findings in the brain vary greatly; from negative ones to such involving the most profound structural changes. I exclude from consideration those exceptional cases where microparasitic invasion, multiple cysticerci, and ulcerative endocarditis were found. These cases seem to illustrate the fact that any multiple irritative lesion, suddenly overwhelming the brain functions, can produce delirium and coma.

In that group of cases due to emotional causes, alcoholic excesses, insolation, and the development of pre-existing insanity into this form, it may be broadly stated that in those terminating fatally before the seventh day, gross lesions of the brain are not found. In those dying on the tenth to fourteenth day, the cortex is found reddened, the white substance may appear discolored,<sup>1</sup> the leptomeninges appear dull and thick, milky streaks are found along the lines of the vessels,<sup>2</sup> sometimes the pia is slightly œdematous, and under these circumstances the cortex, instead of presenting the rosy tint of injection, may be pale gray or yellowish. In cases of dying after this time, remarkable conditions are found. As far as I can learn, the first mention of these was by myself, in a brief communication to the New York Neurological Society in 1878.<sup>3</sup> I found in a case where all the changes thus far mentioned were most marked, the cortex actually appearing mottled with purple, blue, and crimson patches, that a gelatinous material lay in the meshes of the pia, around several of the vessels. I secured some of this from near the floor of a sulcus, but was unable to discover

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<sup>1</sup> Schüle, "Handbuch."

<sup>2</sup> Krafft-Ebing, "Lehrbuch."

<sup>3</sup> JOURNAL OF NERVOUS AND MENTAL DISEASE, 1879.

any traces of organization in it. It was of a pale buff tinge. Throughout the cortex I discovered a number of small perivascular patches, which retained this color even after the scalpel had expressed the blood. They seemed to adhere to the adventitia. Microscopic examination showed that this material constituted a veritable exudation. It was uniformly found around blood-vessels, stuffed to repletion with blood corpuscles, in stasis. To my mind it resembled fibrin in the hyaline state, being homogeneous or finely punctate. Its contour was in most places distinct, in others, a peculiar condition of the neuroglia was found on the perivascular border. This latter in its basement substance stained with a beautiful pink flush, which gradually lost itself in the contiguous tissue. It was as if the neuroglia had imbibed the material exuded, and this being of a protoplasmic character, had given it a higher stain in carmine. In some instances the exudation was enormous, equalling in diameter, on either side of a larger arteriole, from one-half to two-thirds the thickness of the latter. It was particularly massive near the bifurcation, or ramification of the vessels, at the borders of the white and gray substance, and in one single section, fibrin in layers was found firmly united to the neuroglia on the one hand and connected with the hyaline material on the other in three places. There were no pronounced changes in the nerve elements themselves. The nuclei of the neuroglia were about twice as numerous as usual, and collected around the smaller capillaries. The pyramid cells stained poorly, but their processes were well preserved as far as could be seen.

In his lectures on mental diseases, Clouston,<sup>1</sup> failing however to recognize the clinical character of his case, describes small, pellet-like bodies, the size of pin heads and of a glistening appearance, distributed throughout nearly the entire cortex of the brain. His plates<sup>2</sup> show a condition very much like that found in my case. He describes them as found in single spots, or immense lobulated

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<sup>1</sup> Lectures, page 194.

<sup>2</sup> Plate III., page 193, and Plate VIII., page 426.

masses, with a nucleus in the centre of each, quite visible to the naked eye. Probably what he means by "nucleus" is the blood-vessel which runs in the axis of these masses. He compares it to waxy material, and speaks of it as a chemical product deposited around "nuclei." As I have found brains of sufferers from delirium grave peculiarly liable to that dissociation in alcohol which leads to the precipitation of leucin spheres, it is just possible that Clouston has incorporated with his correct observation of the masses found in the recent brain, that of artificial bodies produced in hardening, an error of which Fürstner<sup>1</sup> accuses Yehn with some justice.

More recently Fütterer<sup>2</sup> discovered a large number of yellowish foci, fifteen of them being macroscopic, all located in the subcortical white matter, and which he interprets as the nutritive results of thrombic stasis.

All these changes are, in my opinion, collateral results of the hyperæmia which is the characteristic of the disturbed brain circulation, and throw no direct light on the essential pathological foundation of grave delirium. That morbid changes from thrombic stasis are apt to be most marked in the subcortical white matter, I pointed out in 1877 in explanation of the peculiar disturbance of mental association found in parietic dementia.<sup>3</sup> To explain the phenomena of the former disease, in accordance with established etiological facts, we must assume that in some cases there is a slow, in others a rapid formation of a toxic agent in the nerve centres themselves, a self-intoxication, so to speak. We know that mental states influence the secretions and excretions, both quantitatively and qualitatively. The icterus of sudden rage, jealousy, and fear is no fable. The venomous character of the bites of infuriated human beings and animals are attested by well-observed instances,

<sup>1</sup> Archiv für Psychiatrie, xi., foot-note p. 528. Remarkably enough the error of which Fürstner accuses Yehn, was repeated by a pupil of Fürstner's in a monograph prepared under Fürstner's supervision, based on specimens prepared in his laboratory.

<sup>2</sup> Virchow's Archiv, 1886, cvi., p. 579.

<sup>3</sup> "Psychological Pathology of Progressive Paresis," JOURNAL OF NERVOUS AND MENTAL DISEASE, July, 1877.

and recently I have obtained a ptomaine of intense virulence from the saliva of a dog, dying two months after artificially induced brain disease, in convulsions.

There are features observed in the morbid anatomy of grave delirium which point in this direction. So strongly do they point in this direction that Briand-Marcel examined the blood of seven cases for bacilli, and claims to have found them in three. Fürstner made the interesting discovery, which I have confirmed in one case, that the blood removed from the finger during life is surprisingly dark, and, contrary to what is the rule with dark blood, coagulates rapidly and *en masse*. In addition, the muscles are of a dark brown color, dry, amyloid degeneration is common and most marked on the right side. This change in the muscles is identical with what Zenker, Bowman, Waldeyer, and Wedl found in typhus, variola, pneumonia, puerperal fever, epidemic meningitis, traumatic and inflammatory changes resulting from injury to the muscles, and in tetanus. Either the diminished nutrition or the prolonged muscular strain is the cause of acute delirium. Perhaps both co-operate. Certainly, from the preponderance of this amyloid degeneration on the right side, in two cases observed by him, Fürstner is justified in regarding the latter as playing some part.<sup>2</sup>

This theory, the production of an autochthonous nerve poison, is the only one which accords with the following facts:

1st. That an hereditary or acquired disposition to insanity and other nervous disorders, involving undue biochemical mobility of the nerve centres, exists in over ninety per cent of the cases recorded.

2d. That insolation, alcoholism, and emotional overstrain are the usual exciting causes.

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<sup>1</sup> Archives de Neurologie. 1883. Rezzonico (Archivio italiano per le malattie nervose, xxi., 5) found micrococci emboli in one case; I found perivascular nodules and subpial invasion zones, whose periphery was crowded with microorganisms, whose exact nature the imperfect preservation of the specimens prevented my ascertaining. Briand-Marcel's culture attempts failed.

<sup>2</sup> Archiv für Psychiatrie, xi., p. 530.

3d. That changes in the blood and other tissues, analogous to those found in zymotic diseases, occur.

The lucidity exhibited *sub finem* in some instances, and in the course of the disease at others, is one of the most difficult facts to explain; but it is not without its analogies. Many of the deaths from strychnine poisoning and tetanus recorded occurred, not in convulsions indicating the intense action of the toxic agency, but in a quiescent state, from oblongata paralyses, or other sudden asthenia.

The crucial test, as to the existence of such an agent in the circulation, I made on three rabbits. This was prior to the time when I recognized what a miserable test of the virulence of ptomaines or bacteria this animal is.<sup>1</sup> All the animals died within forty-eight hours, in convulsive stupor, with cries of distress in two instances, in paralytic coma in the third. For the reasons indicated, I attach no importance to these results. I am inclined to attribute more weight to a therapeutical observation, which first directed my suspicions to the existence of some chemical agency baffling treatment. I have given morphine in every legitimate dosage, and even passed what many would regard as the limits. In no instance have the pupils, the pulse, the secretions, the subjective sensations, or the mental state been affected in any way by this ordinarily so powerful and constant alkaloid. I trust that those who are better chemists than I am, and have a larger material at their disposal, will direct some attention to the very important question of the ability of the predisposed and over-burdened nervous centres to poison themselves.

#### REMARKS ON DR. SPITZKA'S PAPER.

The President, DR. GRAY, of Brooklyn.—I understood Dr. Spitzka to say that all the cases were fatal. I have seen two cases of this kind, in everything like those by Dr. Spitzka except the fact that they were not fatal. They had all the phenomena on

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<sup>1</sup> They were terebrated. Fritsch has recently, in the course of his control experiments on *lyssa*, had a similar experience.

which one would base the diagnosis of grave delirium. In one case, the patient went to a lunatic asylum where the diagnosis of general paralysis was made. One patient lived six months or more, and died of intercurrent disease. In one case, there was a syphilitic history, very distinct, of the year previous, but no distinct connection between it and the outbreak of mental symptoms was made out. It seems to me that there are forms, therefore, of what clinically we would diagnosticate as acute or grave delirium, which are capable of passing into the subacute or chronic condition, and, therefore, it is difficult to make the diagnosis. These two cases had these distinct characteristics, which I would ask Dr. Spitzka if he has seen. While they were violent, and the hallucinations were cyclonic in character, they were easily controlled without mechanical restraint. I recollect one case which I put in charge of a trained nurse, and at my next visit I found that the nurse had become demoralized, and had called in a man to assist her, and yet I could stand at the foot of the bed of the patient and get him to lie down or do what I wished. Other cases of grave delirium have not had that characteristic, and there has been no means of preventing them from injuring themselves except by restraint.

DR. C. K. MILLS, of Philadelphia.—I recall the case which Dr. Spitzka mentions in his paper. This patient recovered after a long time. The treatment varied from time to time, consisting chiefly of packs, hyoscyamine, bromides, chloral, administration of food.

I remember three cases, in which I was called in consultation in private practice; three women. All three had been diagnosed as cases of hysterical insanity. Two of these three patients died, and one recovered. I believe that two of them were fairly cases of typho-mania or grave delirium. I mention these cases because of the practical point of mistake in diagnosis, and I speak of fairly calling them grave delirium, because I appreciate the difficulty of drawing the line between typho-mania and acute mania, and perhaps several other affections. I think it is likely that most of the cases of recovery may not have been entitled to the designation.

I noticed, within a week or two, in one of the journals a rather interesting observation, in which a physician in the West very promptly relieved a patient by administering a large dose of morphine and following it with chloroform.

With reference to the more scientific point in Dr. Spitzka's

paper, that of self-poisoning in these cases, although it is interesting, it is one which does not admit of discussion at this time.

DR. SPITZKA.—I cited Dr. Mills' case because there was no question in my mind that it was one of grave delirium, although the recovery was exceptional on account of the readiness with which nourishment could be administered.

With regard to the case treated with morphine and chloroform, I should have serious doubts unless it had been carefully analyzed. These cases do not cease suddenly, and I doubt if it would be proper to administer chloroform.

I must congratulate Dr. Gray on his experience in having seen two cases recover, which is exceptional indeed. He says that some of these patients are quite reasonable with the physician, and I think that in censuring the attendants he is apt to overlook one point, and that is, the patients will show less respect to those continuously around them than to those who come in occasionally. In theory I am opposed to mechanical restraint, but I should find it difficult to get along without it in these cases. Chemical restraint fails in these cases, and that form which would be efficacious would be unjustifiable. The principle of trying to get along without restraint is correct, of course.

I would say with regard to the self-intoxication theory that it is merely suggestive, and we must know when and what to look for. I exclude all acute maniacal cases, and limit myself to those cases where emotional disturbances, alcoholism, and sunstroke are the exciting causes of insanity.

DR. HENRY HUN, of Albany, read a paper entitled,

"GLIOMATOUS HYPERTROPHY OF THE PONS."

Gliomata of the pons are very rare, only a few cases being on record. The following case, occurring in a girl six years of age, showed steadily increasing inco-ordination of movements, bulbar paralysis, and general motor paresis. The father died with symptoms of melancholia and dementia. Two months ago, the patient had an attack of croup followed by a cough. Every time she coughed she felt a severe pain in the top of her head, but at no other time. About three weeks ago she began to walk badly. Her mind is clear, memory good, and she is not nervous. She has an excessive appetite. She is a well-nourished, in-



telligent girl, but has a vacant expression. Her speech is drawling. She keeps her mouth open most of the time, drools when eating, and has some difficulty in swallowing. Her head is drawn towards the right shoulder most of the time, especially when she makes any exertion. She stands with her feet wide apart and is careful not to lose her balance. In walking, she tends to walk in a circle, always turning towards the right. The gait is unsteady, swaying and pitching. There is no disturbance of sensibility in any part of the face, body, or extremities. The plantar reflexes are normal; no ankle-klonus; knee-jerk exaggerated, especially on the right side. Well-marked optic neuritis in both eyes. Urine contains neither sugar nor albumin. Later, there was ankle-klonus, strabismus, and slow panting respiration. Hearing and cutaneous sensibility remained unaffected; sight but slightly impaired. No convulsions. Death occurred suddenly with consciousness to the last.

*Autopsy.*—The bones of the skull were thin. The pons Varolii was greatly enlarged, to three or four times the normal size. On section, it was found to be replaced entirely by a tumor which preserved wonderfully the normal appearance, so that it looked like a greatly hypertrophied pons. A little posterior to the middle of the pons on the right side was a spot of softening, about three-fourths of an inch in diameter. The tumor seemed to be confined pretty accurately to the pons, the crura cerebri and medulla being but slightly enlarged.

Microscopical examination revealed the growth to be gliosarcoma. The tumor produced no symptoms of irritation; there were no convulsions and but little headache. There was simply a steadily increasing loss of function of those nervous elements which are subjected to the pressure of a growing tumor. It was remarkable that, notwithstanding the great amount of œdema of the brain present in this case, and an internal hydrocephalus so extensive as to cause a perceptible enlargement of the head, consciousness, even intelligence, was preserved up to the end of life.

## REMARKS ON DR. HUN'S PAPER.

DR. M. ALLEN STARR, of New York.—I would like to ask Dr. Hun as to the condition of the lemniscus. In the record, he speaks of the symptoms of inco-ordination with ataxia, and I think it is well proven that that symptom is produced in lesions of the pons by involvement of the lemniscus. I would also inquire whether there was ascending or descending degeneration of the lemniscus.

DR. HUN.—The entire pons is very uniformly infiltrated with these new-formed cells, and it is difficult to say that one point is involved more than another. The lemniscus does not seem to be more affected than any other part, and yet it may be.

DR. SPITZKA, of New York.—In gliomatous infiltration of the pons, the lesion might be described as total in some parts and partial in others, with the fibre tracts undisturbed or not, softening, etc., and I think that it would add greatly to the value of the record if that were supplied in the form of a chart. The location of the patch of softening I see is quite distinct.

DR. M. ALLEN STARR, of New York, then read a paper on

PARAMYOCLONUS MULTIPLEX, WITH THE REPORT OF A CASE.<sup>1</sup>

DR. E. C. SPITZKA, of New York.—Whether we are justified in drawing any sharp line of demarcation between this and other conditions is a matter of doubt in my mind. I will add a case of paramyoclonus multiplex observed in my consultation practice, which will bring the number up to eleven.

Adolf L., aged 30, born in Poland, and a saddler by occupation, while working for Brewster & Co., was attacked by a pulmonary affection. On going home he felt a sharp pain over the sternal region, fell down, and had a hemorrhage from his mouth accompanied by cough. Friends took him home and a physician who was called in pronounced the case one of "pneumonia, gastric catarrh, and trouble in the back." He suffered with the disorder thus designated about four weeks; fever subsided, but an intense acute pain was developed in the left pectoral region, for which Dr. Mandelbaum gave him two hypodermics of morphia with the patient's knowledge. He denies that the knowledge of this administration had any great effect on his mind. His own account is as follows: "The morphia was scarcely in five minutes before

<sup>1</sup> See JOURNAL OF MENTAL AND NERVOUS DISEASE, vol. xiv., p. 416.

my arms began to work, they shook violently as if they were steam-pistons crossing each other. This spasm continued four weeks in the right arm, and a year and a half in the left." As I saw this spasm myself in the second attack, I can describe it. The motion consisted of a rapid clonic spasm, the forearm being flexed on the arm, with the palms towards the chest, and in this position both upper extremities underwent a violent clonic sawing motion in the shoulder joint; the symmetry of the motion was remarkable, the intensity seemed to be greater in the left, where it was accompanied by beating of the hand against the right infraclavicular space; the right being crossed over the left, the forearm of the former beat against that of the latter, occasionally striking the left infraclavicular space. At that time he had spasms similar to those to be described, and as at present most marked in the right leg. He recovered after eighteen months, the left arm being the last site of movements of a morbid nature, and worked at his trade of a saddler for nearly six years without an unpleasant symptom. Eight months before my being consulted by Dr. Price, he had an attack of pleuropneumonia with pleuro effusion; at this time his left arm experienced a drawing sensation, and it was spoken of to Dr. Price's predecessor. The patient begged the physician to "do nothing for it," as he feared that no remedy would avail him, even if it did not make matters worse. The physician said: "I will show you that I can do something," and gave him a pill. The patient claims to have become worse, the sawing motion of the left arm recommenced, and the drawing sensation passed to the right. The medical attendant then attempted to render the arm immobile with a bandage. Although this happened five months ago, the arm still shows black and blue marks attributed to that appliance. The cough with which the patient was affected gave place to a peculiar outcry which occurred at the height of the spasm from one to six times, it was "nay," or "hay," or "hirr."

After the left arm the movements on this occasion involved the right leg, and at the commencement of the spasm only these parts experienced drawing sensation and spasm. But towards the acme of the spasm, the symmetrical movements were executed by the other side, the entire trunk became involved, the face markedly so, and the bed and the entire room shook with the violence of his movements. There were a number of points, contact with which evolved the spasms. The most sensitive was on the right knee.

I experienced the same difficulty in testing the knee-jerks mentioned in the other cases of Friedreich's symptom. Mental impressions provoked the spasms and my visit in the capacity of consulting physician provoked an unusually severe one. He called out "nay," and "hay," eight times in twenty-five minutes; the spasm, with occasional ups and downs, lasting that long. I estimated the movements at 140 a minute. I examined him on February 25th the first time and found the pupils equally and greatly dilated, reacting sluggishly to light and accommodation; three days later the left pupil was noted to be distinctly narrower. In the periods of rest, the tongue shows a fine tremor; during the spasm, it moves in and out at a rate synchronous with the movements of the left arm. There are no movements during sleep. It can be distinctly seen that the patient struggles against the movements, perhaps delaying, but ultimately overcome by them. Another person might succeed for a time in checking the incipient movement of the right leg, but if he did so the movements began in the left arm with great intensity and then rapidly involved the whole body, and on the first spasm observed, the left side of the face seemed to precede and exceed the right, but close observation of later attacks showed that both were symmetrically involved.

The cutaneous pressure sense is bad in some parts, particularly on the calf of the leg, pain sense is abolished, a needle having been repeatedly run through the skin of the calf of his leg without being felt. The temperature sense is perfect. He could not distinguish salt, sugar, or pepper five months ago. In a few weeks Dr. Price ascertained that these articles tasted like sand when the patient's eyes were closed, but when they were open, the patient claimed to recognize their flavor. When I tested him he could tell the various articles employed, with his eyes shut, and correctly located the part of the tongue to which they were applied. If he closed his eyes while standing, he would be thrown off his feet violently by trunk spasms.

On June 13th the patient who had been placed on the bromides in large doses, came to my office, a feat previously impossible. He could walk without assistance, and the movements were ordinarily limited to the left arm. As he undressed for examination, a peculiar movement developed in the lower extremities, like the clonic tremor which seizes a man's arm when attempting to hold out a dumb-bell after tiring of the muscles, these gradually raised him up as if stiffening like a strychninized animal, and, but, for sup-

port he would have fallen over in this clonic symmetrical spasm of the trunk, thighs, and legs (?). A careful test of cutaneous sensation revealed no anomaly whatever. Sometimes there is subjective sense of heaviness in the limbs. The pupils were equal and reacted well. Ankle-clonus is easily elicited on the right side, but not on the left. When he closes his eyes the left arm makes a violent turn outward and backward in the shoulder-joint, so as to unbalance him. During the past four months he has improved whenever the bromides were pushed, and deteriorated when they were suspended. On the latter occasions, pain was felt in the back from the neck down, so that he could not lie on it. It was also noticed that a dry cough which recurred was worse when the bromides were suspended.

The patient is a zealous workman of prepossessing countenance, and there is on his part every effort and intention to get well. He always was an excitable man, "very tenacious of what he regarded as his rights, and intemperate in maintaining them." Of four children, one died in convulsions at the age of nineteen months, three years ago. The rest are healthy. Dr. Pierce states that morphine has a bad effect on his heart, and on repeating a trial at my suggestion, he found, and I found that it made the patient distinctly worse, although it was given disguised. I tried suggestion *à la* "myriachit" with no result whatever. Galvanism failed to exert any good effect.

From the character of the movements, their symmetry, the muscles mainly involved, and the absence of signs of organic disease, I believe that the case can be ranked only with the symptom-group described by Friedreich. At the same time, it is noteworthy that the movements were not symmetrical in their severity, and that there were other anomalies, notably the peculiar exclamation, which would seem to indicate that until more cases were observed it would be premature to insist upon a narrow demarcation.<sup>1</sup>

DR. C. L. DANA, of New York.—The patient mentioned by Dr. Starr had one of his convulsive attacks in my office, and I had an opportunity to study it in comparison with a case of convulsive

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<sup>1</sup> The history here furnished is inserted by permission, not having been fully stated at the meeting. Another case, Ernest S. W., the record of whom has since been found, had the typical condition in the trunk and lower extremities, elicitable on pressing the sacro-coccygeal articulation; the assigned cause in his case was a lumbar strain caused by moving a piano. The patient has been seen by other members of the association.

tremor which I had seen nine months before. My case occurred in a boy 17 years of age, and it seems to me that it was essentially the same kind of disease as has been described by Dr. Starr.

With regard to the nature of the disease, while I think it is desirable to draw sharp distinctions when we can, I do not see by what right any one can set up a certain group of symptoms and say that everything which does not show these symptoms has some other disease. Friedreich has not discovered its morbid anatomy or pathology. He cannot say that the symptom-complex is a thing by itself. My own opinion is that convulsive tremor is essentially identical with paramyoclonus multiplex. I think that Dr. Prichard's case goes to show this. I do not think that we can exclude a disease because the wrists are affected, when Friedreich describes a disease in which the muscles of the body and arms are involved.

DR. GEORGE W. JACOBY, of New York.—I believe also, with Dr. Dana, that we cannot draw the line sharply as Dr. Starr has done. I found in my notes the record of a case which I saw in 1884, and which I think will come into this category.

On September 10th, 1884, I saw Mr. W. S., 25 years of age, German, a machinist, and a resident of Hoboken, N. J. I made the diagnosis of "myoclonus spasmodica." My notes then were as follows: Family history unimportant. He had three brothers and two sisters, all well. The patient was well until eight months ago, when he had pain in the sole of the left foot and the pain was severe and momentary when any unequal pressure was made upon the sole, as in walking upon cobble-stones. He then had similar pains on the opposite side, which lasted for five months. He was otherwise well. About three months ago he noticed that when he would stand at work, his knees began to tremble; this was very slight and lasted only a short time. Then his eyes became affected. Everything he looked at seemed to dance before him, so that he thought his eyes were moving to and fro. He asked his friends if they could see anything within, but always received a negative answer. At this time, the tremor in his legs became more jerky, and he had difficulty in walking. Then his head also began to jerk forward and backward, never sideways. He has fallen in the street on account of these contractions in the neck and legs. He is always perfectly conscious. He never had this trembling when he ate or drank, only when standing or

walking; when sitting he was perfectly quiet. The patient is tall, robust looking, talks slowly, and seems to have difficulty in pronouncing words. When he sits still, nothing out of the way is noticed; but as soon as he gets up, his head is thrown forward and backward by clonic contractions of the muscles. These movements began at the rate of about five a minute, but gradually increased up to sixty when he is obliged to sit down, and they gradually cease. Both arms also show short quick movements of flexion and extension, the fingers being continually flexed. These movements are not synchronous with those of the head, and this is noticeable in the beginning when the movements are slow. The muscles of the neck, particularly the sterno-cleido-mastoidei and also the biceps and triceps, can be seen and felt to contract. All voluntary movements can be easily executed. Patellar tendon reflex enormously exaggerated. Slight lateral nystagmus. When standing, if he has the slightest hold upon anything, the movements decrease in intensity. Pressure upon the soles of the feet, when the patient is sitting, also produces the contractions.

While this case does not fall entirely within the description given us by Dr. Starr, yet I think it must be considered as one of the same or a very similar class.

DR. F. X. DERCUM, of Philadelphia.—I also agree with Dr. Spitzka, and Dr. Dana, and Dr. Jacoby, and cannot see any special reason why a fixed description should be adhered to. It seems to me that we must look upon the disease in a broader light, and that these convulsive phenomena have a common cause. Some of the gentlemen present may be aware that with Dr. A. J. Parker I performed some experiments and artificially produced convulsive movements, and we explained the phenomena on the ground of strain at the time and temporary nervous exhaustion. The movements were not limited to any one part of the body, but any part being placed under strain would be affected more violently than other parts.

DR. C. K. MILLS, of Philadelphia.—I must agree with the other gentlemen, although I hope Dr. Starr's equilibrium will not be disturbed. With reference to making this a permanent affection, this symptom-group, I do not see any good reason for separating it from convulsive tremor or from some affections which we call hysterical. It would be interesting if Dr. Starr would give us the differential points between this affection and certain hysterical motor seizures.

I have described two cases somewhat like those described this afternoon, although not so well described and in such detail. I have seen others which are sufficiently close to be placed with them. Only last week a patient died of pneumonia at the Philadelphia Hospital, and in many respects the case was much like those described this afternoon.

H. P., aged 24, colored, born in Pennsylvania, married, was admitted to the nervous wards of the Philadelphia Hospital, October 10th, 1886. She had had measles, whooping cough, and other diseases of childhood, but otherwise was healthy up to the age of 15, when menstruation began. The patient then commenced to have paroxysms of severe headache, usually coming on from four days to a week before the flow, and gradually disappearing as it subsided. She was married at 17 years of age, being about two months pregnant at the time. The child was carried to full term, but only lived three weeks. During pregnancy, her headaches continue increasing in frequency. She had three other children one year apart, the second of which was still-born. She said that the headache was at the vertex and below the eyes. She was always constipated before her admission to the hospital, sometimes going a week without a movement of the bowels.

Her nervousness dated from the birth of her still-born child, and began with pain and weakness in the lumbar region. At times her elbows and knees were very weak, sometimes suddenly giving way beneath her when she was holding anything. This weakness gradually became worse.

She began to have twitching of the arms, trunk, and legs, also of the neck and face muscles. When she remained quiet sometimes in the daytime the twitching ceased. At night, however, the most violent attacks of twitching occurred. Upon touching her slightly, but suddenly, violent spasmodic writhing took place. Any sudden noise had the same effect. She said that she neither felt pain nor fright, but the movement was entirely involuntary. She made a jerking convulsive movement in picking up or putting down anything, but once grasped she held it firmly. Reading caused spasmodic movements of the head and neck. She was a little near-sighted. She could feed herself, and after holding a spoon for a few moments she could carry it to her mouth without spilling. All her muscles twitched upon first touching anything, but upon prolonged touching or holding, the movements ceased. The knee-jerk was very much exaggerated for a few seconds, but



after repeated or continuous trials it was lost entirely. Upon tapping to bring out the knee-jerk, spasmodic movements of the back and thighs occurred. She had pain in the back at times, not marked, however.

This patient's movements were bilateral ; they were symmetrical at the time of the occurrence of the movements ; ordinarily the movements were confined to the legs and the trunk. If I attempted to have her stand they were much exaggerated, yet if she persisted in the attempt she could be made to walk quite well. With all this she had nothing of the condition of tonic spasm of the muscles, and there was no possibility of the case being one of spasmodic tabes, or any condition of that kind.

There is a patient who comes occasionally to my service at the Philadelphia Polyclinic whose record I will also give.

T. T., aged 4 years and 8 months, was sent to the Polyclinic for report as to electrical condition, and both upper and lower extremities responded to the faradic current and galvanic current. His mental powers were deficient. The child had a flat face, with a chronic eczematous eruption. He had a vacant expression and has no speech except to say papa and mamma. His limbs were thin. He was extremely restless and irritable. If he was touched suddenly, or if he heard a loud noise, he suddenly became convulsed without loss of consciousness. His head and body were tossed violently backward, his legs and arms being agitated in the same manner, being tossed in the air in a succession of rapid clonic regular movements. Although the description is very meagre, still it covers the ground.

DR. B. SACHS, of New York.—I would like to say a few words in support of Dr. Starr's position. The term paramyoclonus has only a clinical significance. It does not describe a distinct morbid entity. The fact that we have a large number of functional tremors does not prevent us from making a subdivision and giving it a distinct name. While every one of us will allow that it is fair to speak, of muscular dystrophies, a certain number of subdivisions may be accepted, even if they have a resemblance. There is occasion for distinct subdivisions, in the same way, in this class of cases. The agreement between all of them is very marked ; whereas the distinction between these cases and convulsive tremor, and those of a hysterical functional type is very great. If we reason in that way we need not discard the term, and there is a sufficient number of cases to be entitled to that designation.

DR. WHARTON SINKLER, of Philadelphia.—I have seen a number of cases of hysterical convulsive tremor which closely correspond to the description given by Dr. Starr and others. One in particular I have seen recently, in which there was bilateral convulsive tremor in a girl who was distinctly hysterical. It seems to me that in the cases described by Dr. Starr the hysterical element is more or less present. They all tend towards recovery.

DR. STARR.—I think that the discussion has been profitable; in fact it has appeared that we have all seen cases which do not correspond to any other designation than that put at the head of this paper.

With regard to Dr. Spitzka's case. He says that the patient gave an expiratory sound, and spoke of that as not having been observed in other cases. I mentioned the fact that in my case there was clonic spasm of the diaphragm occurring in almost every attack and resulting in a long inspiration accompanied by a sound. I would also mention that the making of a sound has been recorded in another case. I would also call attention to the fact that, in Dr. Spitzka's case, the spasm was *bilateral* in particular sections of muscles, but involved one side more than the other. In paramyoclonus, the spasm is bilateral and not unilateral. If that is so, Dr. Althaus' three cases and Dr. Pritchard's cases must be ruled out. If they are convulsive tremor, they are not paramyoclonus.

With regard to Dr. Dana's statement that these cases are probably convulsive tremor, I supposed that that would be brought forward, and have therefore brought notes of all the cases described by Hammond and will read them briefly.

It can be seen from these that not a single one of the cases recorded by Hammond, excepting those mentioned in the paper, in any way corresponded to the nature of the disease as made up from the ten cases already described. In Hammond's cases there were marked cerebral symptoms, headache, vertigo, and mental excitement; the spasms were not all of the nature of those in paramyoclonus, the limitation of the spasms was not mentioned, and there were motor and sensory symptoms. The same was true of the case of Pritchard mentioned by Hammond.

I have no desire to multiply diseases, but it seems as if here was a group of clinical cases, resembling one another in many respects, and differing from all other groups of cases, and hence requiring both recognition and a name. Freidreich was the first to record a

case of this kind, and the name selected by him was sufficient and should be adopted. I have no theory of the disease to urge, as data for a theory are not yet at hand. My paper had for its object to bring the subject to the attention of the profession and to record a well-marked case.

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*Friday, Third day, Last session.*

The Association was called to order by the President at 10 A.M.

The report of the Committee on Revision of the Constitution and By-laws was read and discussed, and laid over for one year under the rule.

DR. E. C. SPITZKA, of New York, from the committee appointed by the President, asked permission to furnish a minute on the death of Dr. Thomas A. McBride, to be entered in the published transactions. It was granted. (Dr. Spitzka furnished the following memorial note):

Dr. Thomas A. McBride was one of the earliest and most active members of this Association. It is to be regretted that so little of his work is recorded. He was one of those who hesitate to publish, until convinced beyond a possible doubt that their observations are at once novel, useful, and incontrovertible. In this way, many an original clinical study remained buried in his drawer, having been made subsequently elsewhere, anticipating his too long delayed publication. The impress of his mind was, however, liberally affixed to the work of others. The members of the active and progressive circle to which he belonged, were in the habit of consulting him regarding many of their experimental and clinical researches. His advice was always freely and unselfishly given, as his magnificent library, and large armamentarium of instruments were always at their disposal. As might be anticipated from one who was so extremely cautious in publishing his writings, Dr. McBride's contributions to clinical neurology were of the very highest order. They related, in large part, to the use of instruments of precision, and to the analysis and differential diagnostic significance of special symptoms. Some of the best monographs on the latter subject were published as editorials in the *American Jour-*

*nal of Neurology and Psychiatry* of which he was the founder, and which was discontinued owing to his disability from the disease which ultimately ended his life. It is a melancholy reflection for his surviving friends, that for many years, during which he was recognized as the foremost authority on Bright's disease of the kidneys, on gout and gouty states, he was himself suffering martyrdom from both, and successfully concealed the fact from most of them. The disposition to these diseases he inherited, and his father followed the son to the grave, a few weeks after the latter's death from the same combination of these maladies.

As regards Dr. McBride's personal qualities, language seems entirely inadequate to portray the unselfish devotion, disinterested loyalty, and kindly generosity which he uniformly manifested towards his friends. He seemed to penetrate so thoroughly the innermost personality of those who were privileged to be his intimate associates, that at this day it seems almost impossible to them to realize his death in all its stern reality.

He left with kindly greetings to all of us, knowing that disease had made such inroads on his system, that a return home was doubtful. In large part, his journey was undertaken to supervise the treatment of a number of his private patients, sojourning at Carlsbad. One of them related to me the unselfish devotion to their interests which he showed even at a time when it was no longer possible for him to conceal his sufferings. He knew his fate and met it philosophically. Taking a sphygmographic trace one day at Tunbridge Wells, where he stopped on his return, and the patient asking him the object of the instrument, he took his own tracing in her presence, and said, "yours shows nothing of any serious nature, but you see there is a difference between that one and this one—it is my death warrant, I must leave immediately, if I am to die at home."

At Southampton, while awaiting the steamer that was to take him home, he had two uræmic convulsions, and being removed to the steamer in consonance with his request, slowly sank into coma, and died on the second day

out. He was surrounded by a number of the profession of New York City at the time, as well as by other friends. Every effort was made, every sacrifice promised, to induce the Captain to attempt the bringing of his body to New York. But various obstacles interfered, and he was buried far away from his home in Ohio, on the very spot indicated by the last mark of his busy pen on the ocean chart which he had conned in his last conscious moments; his thoughts impatiently winging their way in advance of the vessel, to the land which he was destined never to behold again.

His profession has lost a member who reflected high honor on it, at home as well as abroad; his aged mother lost one of the best of sons; we have—all of us—lost one of our noblest associates; some of us, our best friend!

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The Committee to whom was intrusted the preparation of a brief memorial of Dr. James Stewart Jewell, feel that in his death the American Neurological Association has met with its greatest loss during the thirteen years of its existence. He was one of the founders of this Association, and was its President for the first three years of its existence, from 1875 to 1878. He was always deeply interested in its success, and regularly attended its meetings and took part in its proceedings, until failing health rendered this impossible.

Dr. Jewell was born near Galena, Illinois, September 8th, 1837; he died April 18th, 1887. His early education was limited, but he made up for the lack of opportunity by the energy with which he pursued his private studies. He graduated at the Lind University of Chicago, now the Chicago Medical College; and after practising medicine for a short time in the country, he was appointed Professor of Anatomy, and later Professor of Nervous and Mental Diseases in this College. During most of his life his practice was mainly in nervous and mental disorders.

Dr. Jewell was the founder and first editor of the

JOURNAL OF NERVOUS AND MENTAL DISEASE, and under his management this Journal took a foremost place among medical periodicals, a position which it has since maintained. About one year before his death he founded the *Neurological Review*, but was compelled to give up its publication on account of increasing ill-health. He contributed many valuable practical papers on neurological subjects to this journal and also to other medical periodicals. Probably no man in America possessed a greater knowledge of the literature of neurology and psychiatry than Dr. Jewell. He had by his own exertions become proficient in French and German, and was thoroughly at home in the medical literature of these languages. He took great pride in his library, which was both choice, and large, and in which was to be found almost every recent journal or book of value in the branch of medical science to which he was most devoted.

To other hands will fall the duty of furnishing a complete biography of Dr. Jewell. We, as representatives of that Association of which he was one of the founders, over which he presided, to which he contributed some of his best work, and for which he held a deep affection, simply desire to place on record our high appreciation of his noble qualities of heart and mind. To know him was both to love and respect him. He was of a kind, sympathetic nature, warm in his attachments, charitable in his judgments, gentle in his manners, and highly appreciative of the virtues of his friends.

Some of the older members of the Association will recall the earnest and kindly manner in which he entered into its debates. Always enthusiastic in advancing or supporting his views, he was never personal nor disagreeably aggressive in their expression. Although he did not live out the full measure of his days, he acquired an assured position in his chosen department, and has left behind a name which will not be forgotten by American Neurologists.

(Signed),

CHARLES K. MILLS, M. D.  
ROBERT T. EDES, M. D.

DR. ISAAC OTT, of Easton, Pa., read a paper on

THE THERMOGENETIC APPARATUS.<sup>1</sup>

REMARKS ON DR. OTT'S PAPER.

DR. E. C. SPITZKA, of New York.—As to the clinical experience derived from the study of tetanus, I would direct attention to the report by Dr. Kinnicutt, of a case of chorea in an adult in which the temperature ran up to 104° F. average, reaching at times 107° F. There was no other reason to account for this high temperature except muscular workings. The case terminated in recovery. Other cases were also reported from literature. I have seen elevated temperature in cases of choreic movements, one in which the temperature rose to 103° F., and that case terminated fatally.

DR. C. L. DANA, of New York, read a paper with the title

ANENCEPHALIA ILLUSTRATING THE SENSORY TRACT.

which included the detailed report of a case, in which the corpora striata and both cerebral hemispheres were entirely absent.

REMARKS ON DR. DANA'S PAPER.

DR. M. ALLEN STARR, of New York.—This paper does not admit of a very exhaustive discussion here, because such sections must be carefully examined before discussion can take place. There is one point, however, on which I wish to speak. Fibres were represented in the drawings as being present in the raphe to a greater extent than I could see them in the specimen.

In my case, there were no vertical fibres in the raphe, and that was the reason that I inclined to the conclusion, pointed by some one else, that the fibres of the raphe unite with the nuclei of the cranial nerves. If these fibres were present in Dr. Dana's case, then my view must be given up, for there are no pyramidal tracts; Dr. Dana's case is rather more valuable than my case because it was possible to observe the spinal cord, and furthermore the condition of the corpora quadrigemina and the crura were more distinctly seen than in my case.

I simply wish to present here four specimens prepared by Prof. D. J. Hamilton, of Aberdeen, Scotland, as they are worth putting

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<sup>1</sup> See JOURNAL OF NERVOUS AND MENTAL DISEASE, vol. xii, p. 428.

on record. They confirm in every respect the statements made in my paper, and also to a large extent those made by Dr. Dana.

There were four specimens; one of the pons, one of the medulla, and two of the spinal cord from an anencephalic foetus five months old, born alive. In the pons and medulla there was a total absence of the pyramidal tracts, and the lemniscus appeared to be about one-half its normal size. In the cord the pyramidal tracts were apparently undeveloped, although there was no such indentation on the surface of the lateral columns as appear in Dr. Dana's and in Flechsig's cases.

At this age, in the foetus, the pyramidal tracts in the cord are not yet developed in a normal case, so that it is difficult to judge from these specimens whether in such a condition they might or might not develop. But the cases of Flechsig, Dana, Gretschnikoff and my own combine to prove that in anencephalous brains the pyramidal tracts, which normally develop from above downward, are absent. These cases, with those of Rohon and Hamilton, bring the number of such specimens examined up to six. They are really natural atrophy-experiments according to the method of Gudden and are of much value in tracing the course of tracts through the brain.

DR. E. C. SPITZKA, of New York.—The theory as to the connection of the cranial nerve nuclei with the pyramid tract by means of the raphe is an old one, having been advanced twenty years ago by Meynert. It is certainly established that not even a majority of the raphe fibres have this origin.

With regard to Dr. Dana's case, the one omission I regret is the relation between the thalamus and thalamic fibre fields. I think it would be well to thoroughly review that question.

Dr. Dana's proposition, that in such cases as his the centripetal tracts do not suffer atrophy, is entirely too broad. That portion of the lemniscus which is the continuation of the interolivary layer, connected cordwards with the nuclei of Goll's and Burdach's columns, and designated by v. Monakow as the "cortex-lemniscus," atrophies when the cerebrum is eliminated, whether artificially as in young animals, or by disease. I have published a case of the former kind where all efferent tracts and nine-tenths of the cortex-lemniscus were absent.

In connection with the remarks on the lemniscus, I have brought here a section which shows ascending degeneration of



this tract in the thalamic level. Descending degeneration of the interolivary part has been now described in four cases.

I have also here a section which illustrates what might be called a natural atrophy, and in one sense hypertrophy experiment. It is taken through the enormous post-optic lobes and pons of a porpoise.<sup>1</sup> This animal, having no functional posterior extremities, has no columns of Goll and no nucleus of that column. It has no cortex lemniscus, except the bundle from the pes to the tegmentum be so regarded. The lateral lemniscus, representing the continuation of the trapezium and auditory nerve, is immensely overgrown.

I notice in the diagrams passed round—I think it is marked level 12th to 5th—two areas colored red, as if to indicate ganglionic substance in a region which, if I understand the diagram rightly, contains no ganglionic substance. It seems to occupy the region where the bundle from the pes to the tegmentum runs ordinarily.

DR. DANA.—I would like to say with regard to the drawings simply that I think they are very correct. I made them with a great deal of care.

So with regard to the raphe. I am sure it is well developed in the sections at the level mentioned. At the higher levels it is absent.

With regard to the thalamus, I hope to be able to work it up a little more, but I fear that in this case the thalamus is absent; that is, strictly speaking, it was almost of no account.

DR. GEORGE W. JACOBY, of New York, then read a paper on

#### THE TREATMENT OF NEURALGIAS BY INTENSE COLD.

Since 1884, our methods of applying cold therapeutically have been revolutionized, and this has been accomplished by the introduction of chloride of methyl. Since Débove recommended this agent in the treatment of sciatica, so many cases have been reported that the matter must receive some attention. The author of the paper has reached the conclusion that we possess only two refrigerants which can be easily and practically utilized, and those are the chloride of methyl and the fluid carbonic acid.

The apparatus for using the methyl was exhibited, and the success which he had obtained in its use was sufficient

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<sup>1</sup> It was a Dolphin (*Tursiops tursio*).

to warrant its recommendation, but the difficulty of obtaining it and the expense, were obstacles which had not yet been overcome, but probably could be should the demand for it become sufficiently large. Some reference was then made to the literature of the subject and the reports of cases. From his brief experience, his general impression was, that we have in the chloride of methyl a reliable analgesic which does not affect the general condition of the patient, and that it is invaluable in the treatment of neuralgia for the immediate relief of severe pain.

From his experience in the use of the condensed carbonic acid, his conclusions were that, in the absence of the chloride of methyl, it was able to take the place of that remedy in the treatment of sciatica; that the pain is relieved very promptly by its use; but that its curative effect is not as great as that attributed to the chloride of methyl by other observers.

As to the *modus operandi*, he had come to agree with John Marshall, that in very many cases the neuralgic pain is situated in the *nervi nervorum*, the existence of which have been demonstrated by Victor Horsely, and which are acted upon by the intense cold. Dr. Jacoby, however, was far from believing that the pain in all cases of neuralgia is in these *nervi nervorum*, and consequently in those cases in which the seat of pain is in the nerve fibre itself, that seat not being so accessible to the freezing process, the pain will necessarily return after a longer or shorter interval.

DR. R. T. EDES, of Washington.—I would ask Dr. Jacoby if he has used rhigolene.

DR. JACOBY.—I have, but have abandoned its use.

In the first place it cannot be sent over a sufficiently large surface, and in the second place we do not obtain the degree of cold obtained by these agents.

DR. C. L. DANA, of New York.—I have used rhigolene in the treatment of burning sensations, and the pains of locomotor ataxia. I have only used it in a few cases, however, and I can simply say that in sciatica it has produced decided relief for a long period of time. But it requires a large quantity of rhigolene, and although

it is cheap, it takes so much that the treatment is, on the whole, rather expensive. My observations, so far as they go, confirm the statements made by Dr. Jacoby, regarding the value of cold in the treatment of pain.

DR. CHARLES K. MILLS, of Philadelphia, next read a paper entitled

REMARKS ON POLIOMYELITIS AND MULTIPLE NEURITIS OF  
SYPHILITIC ORIGIN.

REMARKS ON DR. MILLS' PAPER.

DR. F. T. MILES, of Baltimore.—As I shall be obliged to leave you within a few minutes, I would like to say a few words on this subject in which I have been so much interested.

First, with regard to the mixture of multiple neuritis and affections of the cord, since Leyden's paper was published pointing out the frequent error of supposing that we have myelitis when we have multiple neuritis, I think I have seen it several times, and even when both are involved it is difficult to say which commenced first, or to say that they did not begin together.

With regard to the onset, we have many forms which differ exceedingly. Some forms are painful, and others are painless. Some forms seem to pick out the sensory fibres. In some cases I have seen it has been difficult to say at what particular point the nerve was affected, and where there did not seem to be any atrophy of the muscles or degeneration reaction, or at least it did not come on as soon as in other cases. Pain on pressure along a nerve trunk is a very variable symptom in neuritis. There are cases in which we cannot make the diagnosis between central and peripheral paralysis. The best points in these cases are that, if we have paralysis of the muscles with atrophy and degeneration reaction, with loss of sensation, it is most probably neuritis; if we have sensation entirely preserved, it would look like a case of central trouble.

DR. R. T. EDES, of Washington.—I have been very much interested in these two affections. Some who are present may remember that I once reported a case to the Association where there was extensive symmetrical lesion of the anterior columns. The case was one in which there was the usual amount of paræsthesia at the beginning, with rapid paralysis and marked muscular atrophy below the knees and elbows. The case progressed to a fatal termi-

nation. At that time nothing was known about general neuritis, and I found in the spinal cord this lesion. Since that time I have seen a number of cases which somewhat resembled this one, one almost an exact counter-part, but no post-mortem was allowed. That case would have been diagnosticated now as one of multiple neuritis.

I think that Dr. Mills is correct in his assumption that the two diseases co-exist—neuritis and degeneration of the cord. Whether it is as he suggests, the poliomyelitis and neuritis existing together, or whether it is possible there should be degeneration of the cells of the cord as the result of the neuritis, I do not feel quite so certain. My impression is, that the neuritis is diagnosticated very promptly; that is, that there is a tendency now to diagnosticate cases as alcoholic neuritis, etc. That I think is going too far, and I should be inclined to accept the criterion of pain to a considerable extent.

The case Dr. Putnam spoke of yesterday, where there was degeneration of the cord without much inflammatory change, with the sequel of pressure neuritis, is interesting in this connection. In the case I reported, the sections seemed to show to some of the members sclerosis in the white columns of the cord. I examined them carefully at that time and have examined them since, and if there is any sclerosis it is very slight.

DR. J. J. PUTNAM, of Boston.—With regard to the connection between multiple neuritis and affection of the anterior cornua of the spinal cord, I have seen cases which have been pronounced to be poliomyelitis which really were of neuritic origin. I had opportunity to make post-mortem in a case where the pain was intense, came on suddenly with paralysis, and although the nerves were not examined at the autopsy, I concede that they were practically affected, and the spinal cord was the seat of inflammatory process from one end to the other, confined chiefly to the anterior columns of gray matter.

I have seen a case where the symptoms were absolutely typical of multiple neuritis, where the cord was examined and appeared to be healthy, but the brain contained a number of spots of softening.

It has seemed to me, as Dr. Mills has suggested, that the different types of neuritis are so many, and differ from each other so strongly and decidedly, that we have to conclude we are dealing practically with systemic diseases, and that under these circum-

stances the corresponding part in the spinal cord will be found to be more or less involved with the peripheral portion of the nervous system ; or perhaps one case will show peripheral changes alone, and other cases only changes in the spinal cord ; but that there is probably some relation between them.

In a recent number of the *Archives of Psychiatry*, is a paper by Oswald on the subject of lead paralysis, in which he adopts the same view, and speaks of neuritis due to lead confining itself to the motor elements of the peripheral nerves and that the spinal cord is sometimes involved, and that its tunics are affected when no absolute lesion is present, and that possibly and primarily these disturbances of the tunics cause the nerves to suffer.

There is another single point, and that is the matter of absence of knee-jerk, which has been considered as a diagnostic symptom. Several cases have been reported where the knee-jerk was exaggerated in peripheral neuritis, and I can add one or two cases. Whether this exaggeration of the knee-jerk implies a process in the cord is uncertain, and it may be only part of the general condition of hyper-irritability of the nervous system. But the possible error of adopting this as anything like a pathognomonic sign should be borne in mind.

DR. MILLS.—I should like to get the opinions of the members present as to whether or not the presence of tenderness and pain along the course of a nerve trunk with hyperæsthesia can be present, in the absence of peripheral nerve disease.

DR. JAMES H. LLOYD, of Philadelphia.—I would like to say a few words with reference to the electric diagnosis of these diseases. In a somewhat large experience in nervous diseases at the University Hospital, I have attempted to make a differential diagnosis between these two affections, by means of electricity.

I would not draw the conclusions too definite or precise, but it has seemed to me that, in some cases at least, there is this distinction, which I throw out merely as a suggestion.

In neuritis, we get more readily the typical reaction of degeneration than in poliomyelitis ; especially loss of faradic reaction in the nerve trunk, and degeneration reaction to the galvanic current in the affected muscles.

I have experimented with Bell's palsy, and taken it as the type of what I have done in multiple neuritis ; it is not so well-marked perhaps, but where we have established rapidly the typical reaction of degeneration.

DR. M. ALLEN STARR, of New York.—I cannot agree with Dr. Lloyd in this matter of electric diagnosis. I have taken a great deal of pains, in many cases, in making careful electrical observations, and I have tried to test the point made by Dr. Lloyd in his published paper, but I have not been able to verify it, because the cases vary so much in intensity, and I do not think that from a single examination with electricity we can arrive at any very definite conclusions.

I think that Dr. Mills is correct in some of his conclusions, and I have to admit the concurrence of neuritis and myelitis, also neuritis and myelitis and encephalitis, but the point comes up, can any one lesion explain the symptoms if that be the diagnosis? It seems to me that the important point is, to decide whether the cord is implicated in the maximum way. It seems to me somewhat questionable whether pure poliomyelitis often begins with sensory symptoms. I think that one important point is this. I do not think I can say that I have seen any case of poliomyelitis anterior in which the affected muscles were absolutely symmetrically affected upon both sides of the body; one leg will be more involved than the other. A few muscles will be affected in one leg which are not affected in the other; and on one side the muscles will recover more than on the other. That is not the case with multiple neuritis, where the affection is markedly symmetrical. I do not believe that it is policy to call all recoveries cases of neuritis, for there are undoubted cases of anterior poliomyelitis of a mild type which recover entirely, etc. In trying to make a picture of the disease in the lectures referred to, I excluded all cases except those which had been brought to an autopsy, and the number of cases shows that neuritis is not always favorable in its course, although the prognosis is better than in poliomyelitis.

It is fallacy also to say that a gradual onset always means neuritis, for in some cases of neuritis the onset is sudden, for example, alcoholic and lead cases, and in some cases of infantile paralysis the onset is subacute.

It seems to me, however, that pain and tenderness are very important points in making a diagnosis; and they are the points on which I have laid greatest stress in consultation cases. If there is pain or tenderness, not only along the trunk of the nerve, but in the muscles—and in many cases the tenderness is very marked, and both sides are about equally involved—it is a case of neuritis; for these are not present or are very rare in poliomyelitis, so far as

my observation goes. These with irregular areas of anæsthesia I regard as the most important points in diagnosis.

With regard to Dr. Mills' second case, it seems to me that is probably a case of injury; a case of paraplegia developing after injury to the spine, without trouble to the arms. I would like to ask whether there were any microscopical appearances which indicated neuritis in that case. I have just made an autopsy in a case of lead palsy in which the gross appearances were sufficient to make it a case of neuritis, but on microscopical examination there was no marked change.

DR. E. C. SPITZKA, of New York.—I was much pleased with Dr. Mills' propositions which are in the main sound, and equally well pleased with Dr. Starr's admission that the cases recorded are too few to base sweeping conclusions on. The uncertainty in differential diagnosis of peripheral neuritis were aptly illustrated to my mind by Dr. Starr's paper—the one quoted by Dr. Mills—scarcely two weeks had elapsed after I have read it, when case records had accumulated which proved the differential criteria therein laid down to be all or nearly all fallacious.

Within four or five months I have seen three cases like the one reported by Kast in the *Deutsches Archiv für klinische Medizin*, one of them in consultation with Dr. Laurence Johnson. In Kast's case, all the symptoms so accurately aped an acute bulbar paralysis that that diagnosis was made. The patient dying, a careful search was made for cerebral lesion, and none found; the brain, medulla, and pons were absolutely healthy. There was intense neuritis of the nerve trunks whose functions had been disturbed.

In my mind there is no doubt that there has been an extreme tendency to enlarge the domain of peripheral neuritis, attributing obscure disturbances of nerve function to neuritis on the fallacious theory of curability or non-curability. There are fatal cases of neuritis and curable cases of myelitis.

There are scattered records of cases in which even to-day we would look for central disease and where the autopsy showed it, and where the nerve trunks or rather their peripheral expansions were tender. I am therefore not at all convinced that such conditions as Dr. Mills refers to, do not exist.

With regard to the post-diphtheritic neuritides, I would add that it seems to be accepted that they prove the diphtheritic nature of what clinically appeared as a simple angina. It must be remem-

bered, however, that such neuritis has also been recorded as following mumps.

DR. C. L. DANA, of New York.—I have seen cases of multiple neuritis due to alcohol. I believe that alcoholic paralysis is multiple neuritis, and I think that is well established. Certainly, that is almost the rule, without question. In these cases of alcoholic paralysis we find the greatest variety of symptoms, and I have reached the conclusion that it is impossible from objective symptoms to make a diagnosis of multiple neuritis. I place much more reliance upon the fact that multiple neuritis is caused by toxic influences. In alcoholic paralysis we find neuritis alone, and in myelitis we find evidence of myelitis alone; and in a few cases they have been found associated, but these cases are rare. I was very favorably impressed with Dr. Mills' position, and it seems to me that it is very tenable one to take, so long as there have been so few autopsies in which such changes have been found.

THE PRESIDENT.—I do not wish to unduly prolong this discussion, and will only say that I believe the differential diagnosis between central lesion and multiple neuritis to be a very uncertain one.

DR. MILLS.—I did not wish to deny the frequent occurrence of multiple neuritis, but I wished to help on the discussion, so as to give us, if possible, clear distinguishing points between these two diseases. I do not agree with Dr. Lloyd as to the electrical conditions giving any positive indications. From the nature of the lesion in the two affections, we should have the reaction of degeneration in about the same degree.

I am convinced from a large experience, especially in the Philadelphia Hospital, that poliomyelitis does begin sometimes with sensory symptoms; or I believe that the truth is that the cord has been attacked by a more general process which becomes limited in a short time to the horns.

With regard to the special question I asked about pain in the nerve trunks in absence of peripheral nerve disease, it is an important one, and an important diagnostic mark. Take cases of brain tumor; I think most of us will agree that in all but a few cases there are hyperæsthesia and nerve pain in remote places. In certain cases I have demonstrated a tumor at the autopsy and the existence of cortical lesion, and no evidence of peripheral lesion; in all these cases there were hyperæsthetic regions, and



I have recorded hyperæsthesia as a general diagnostic mark in cases of brain tumor.

As to the absence of knee-jerk, no one could contend that it was evidence of multiple neuritis.

DR. E. D. FISHER, of New York, read a paper on

#### BULBAR PARALYSIS

and reported a case which he had observed throughout its entire course, and was able to give the microscopical findings after death.

Mrs. H., æt. 29, family history good, no history of syphilis, several children and no miscarriages. In July, 1885, her oldest son was drowned, which greatly affected her; towards November of the same year her family noticed some difficulty in her articulation. I first saw the patient in February, 1886. She then presented the characteristic symptoms of the disease. Her tongue could only be protruded just beyond the teeth, her lower lip hung down, and the saliva was very freely secreted, running from the corners of the mouth; deglutition was performed with difficulty, and it was almost impossible for her to pronounce linguals and labials. There was bilateral paresis of the left palate, but taste, sensation, and smell were unaffected.

There was no paralysis of upper or lower extremities, and the reflexes were normal. Electrical examination showed reduced response to the faradic current, but the reaction of degeneration to galvanism was not present.

The disease confined itself strictly to the hypoglossal nerve distribution and the lower branch of the facial; the latter, however, not being so severely affected. The patient was shown before the New York Neurological Society in the spring of 1886.

This condition progressed but slowly for the next two months. The tongue finally lay motionless in the mouth, all speech was impossible, it becoming necessary to communicate her wishes in writing. Deglutition became more and more difficult, until the patient became much emaciated and died from inanition January 8th, 1887.

Autopsy was made a few hours after death by Dr. M. A. Starr.

The body was extremely emaciated, with marked atrophy of the lower part of the face and of the tongue. On opening the skull, the dura mater was found closely attached to the inner plate, but no adhesions; the brain substance was pale; no capillary hemorrhages or other lesion, however, present.

On the floor of the fourth ventricle the region over the hypoglossal nucleus on both sides appeared depressed, and the nerves themselves small and of a pale gray, translucent color.

Microscopic examination after hardening in Müller's fluid and staining with carmine and after the Weigert method, showed an almost entire disappearance of the cells of the hypoglossal nuclei, with atrophy of the nerves. There was also decrease in the number of cells in the nuclei of the facial, of the so-called lower nucleus of Clark, or the accessory facial nucleus of Ross, lying in a group between the hypoglossal and vagus nuclei.

The 9th, 10th, 11th, and other cranial nerves were not affected. The walls of the blood-vessels were thickened, and there was some increase of connective tissue.

We have had to do evidently, as the history indicated and as the autopsy confirmed, with a case of paralysis of the tongue and the lower lip of bulbar origin, running its course in about one year and a quarter, and brought on, without doubt, by emotional excitement caused by the sudden death of the patient's son. The character of the case is precisely the same as that of ophthalmoplegia externa or progressive muscular atrophy, differing only in the seat of the lesion.

The changes are induced, most probably, by altered blood supply leading to change in the nutrition of the cells; the strictly limited character of the disease being due to the arterial distribution.

Bulbar disease may be preceded by, or less often followed by progressive muscular atrophy, as in the case of a Mrs. N., æt. 38, under my care for a year

and a half. In this instance progressive muscular atrophy was the primary disease, otherwise the progress of the case has been precisely the same as the one just reported.

These cases are unassociated with any cerebral symptoms, and mark themselves out from the cases of pseudo-bulbar paralysis reported, in that the latter are sudden in their commencement, and are accompanied by hemiplegia.

In cases of embolism, thrombosis, or hemorrhage in the pons with bulbar symptoms, the attack is sudden, usually accompanied with loss of consciousness and hemiplegia or paraplegia. The following case, under my care in the dispensary department of New York University Medical College, illustrates this: Fred. D., æt. 35, giving well-marked syphilitic history, reports that two years ago had an attack of crossed paralysis, involving left side of face and right side of the body, and a month later was affected on the opposite side. One year later, patient had a third attack, in which consciousness was lost for four hours, but patient was not further paralyzed. The condition of the patient, January, 1887, was as follows: patient pronounced linguals and labials with great difficulty; the tone was nasal; the tongue could not be protruded beyond the teeth, and deglutition was almost impossible. There was also, however, considerable hemiplegia of the left side. The case is evidently one of hemorrhage first in left lower portion of the pons, and later on the opposite side, following syphilitic disease of the arteries, involving the hypoglossal and facial nerves.

This case, as others reported, differentiates itself from primary bulbar paralysis by the acuteness of its symptoms, the paralysis of the extremities, and by the non-progressive character of its course.

#### REMARKS ON DR. FISHER'S PAPER.

DR. GRAEME M. HAMMOND, of New York.—I have seen one rather remarkable case of this disease. I exhibited the patient to the N. Y. Neurological Society in 1881, and the disease was then

quite well marked; the paralysis of the lips and tongue. The patient did not die until 1887. In 1884, it was pronounced a case of hysteria by some of the members of that Society, but the patient died with all the symptoms of bulbar paralysis.

DR. THEODORE H. KELLOGG, of New York, read a paper entitled,

#### HYDROTHERAPY IN MENTAL DISEASE.

The term hydrotherapy embraces every form of internal, as well as external, use of water in the cure of disease. The external use corresponds more nearly with the scope of this article. The author gave a brief historical reference, in which he alluded to the Mosaic law, the Mohammedan, the Arabian, the ancient Greek, the Roman practices with water, and it appeared that, during all these ages, cases of insanity, like general diseases, were doubtless treated more or less in the hydrotherapeutic way. This was so, certainly, with cases in France, Italy, and Germany during the eighteenth century.

The most comprehensive principles of guidance for the practitioner in the use of water as a therapeutic measure may be briefly summed up as follows:

First. Careful physiological experiments have established the effects of hot and cold water on respiration and circulation, bodily temperature, the increase of oxygenation and carbonic acid, the conversion of fat, and changes in the nitrogenous tissues.

Second. Rational hydrotherapeutics in mental, as in other diseases, must consist in the application of these physiological facts to meet symptomatic indications in accordance with the etiology and pathology of each individual case.

The author of the paper then spoke of the various forms of baths and of the other external applications of water, and mentioned special cases of mental disease in which they are indicated; the Turkish bath in cases of insanity. It is a vaso-motor stimulant in all conditions of capillary stasis, as found in the bluish extremities of melancholia attonita, primary dementia, and many secondary forms of insanity,

with torpid circulation. An additional advantage of the bath is the passive exercise of the massage, etc. The Turkish bath, however valuable it may be in many affections, is not to be recommended in anything like a routine way. General contra-indications are all organic diseases of the heart and lungs and nervous centres, and yet, in these various instances, a mild degree of dry heat followed by a tepid spray and rubbing are palliative measures.

The Russian bath is an external nervous stimulant, increasing arterial action and diaphoresis, and answering, in the main, the same indications as the Turkish bath.

The Roman bath is a desirable modification of the bath by inunction.

Mention was also made of the hot and cold water baths, shower baths, which should be cautiously employed, packs, douches, mustard baths, ice-caps, rubber coils, salt-water baths, etc.

To sum up the conclusions, it may be said that the indications of hydrotherapy in mental diseases are to control bodily temperature, to stimulate local and general circulation, to produce diaphoresis and the elimination of certain substances through the skin, to hasten tissue change, to improve general nutrition, to allay irritability of peripheral nerves, to procure sleep, and relieve cerebral anæmia and hyperæmia, and, in a measure, to take the place of drugs.

Balneotherapeutics in insanity are employed empirically as yet, but they deserve a much more extended employment than has yet been accorded to them in this country.

#### REMARKS ON DR. KELLOGG'S PAPER.

DR. RALPH L. PARSONS, of New York.—I would like to say a few words in commendation and corroboration of the statements made by Dr. Kellogg in his paper. In the use of different forms of baths, extreme care should be exercised in the use of extreme cold or the higher degrees of heat. In either case, it has been my practice, if the bath is at all prolonged, to apply first a moderate degree of heat, and gradually increase it; and so with regard to

the use of cold, commence with tepid and gradually reduce the temperature.

So also, as mentioned in the paper, shower baths should be used with a great deal of caution, and under the direction of the physician himself.

DR. KELLOGG.—I would say that during several years spent in visiting the asylums in Europe, I found that in England, France, and Germany, general use was made of this means, and I have been surprised that in this country the more general use of water has not been adopted. In the use of this measure we are far behind those countries.

DR. C. K. MILLS, of Philadelphia.—During the last eighteen months this measure has been used to some extent in the Philadelphia Hospital, and also in the State Hospital for the Insane at Norristown, and with great benefit in certain cases. In the cases of grave delirium, the cold pack and baths were used, and proved to be of great value. And the method of using warm baths with cold affusion to the head I have seen employed with advantage in a few cases.

The following papers were read by title:

"The Treatment of Progressive Locomotor Ataxia with Rarefied Air," by H. M. Lyman, M.D., of Chicago; "Hemiplegia in Childhood," by Philip Coombs Knapp, M.D., of Boston; "Illustration of Error in Diagnosis of Some Nervous Diseases," by Irvine C. Rosse, M.D., of Washington; "On the Anatomical and Physiological Relations of the Tract Usually Designated as the Column of Goll," by Nathan E. Brill, M.D., of New York.

#### OFFICERS FOR THE ENSUING YEAR.

*President*, J. J. Putnam, M.D., of Boston.

*Vice-Presidents*, Wharton Sinkler, M.D., of Philadelphia, and B. Sachs, M.D., of New York.

*Secretary and Treasurer*, Graeme M. Hammond, M.D., of New York.

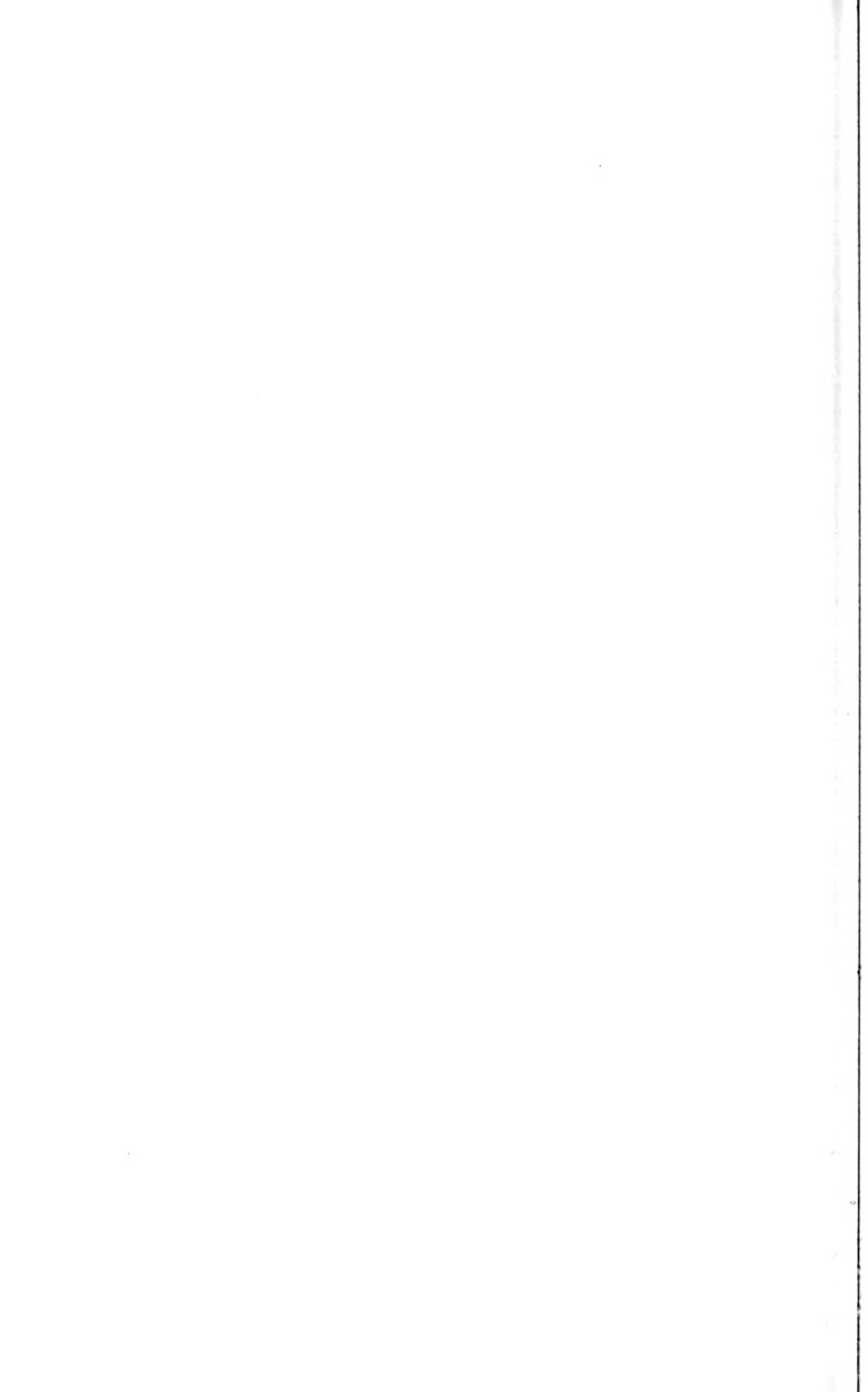
*Councillors*, George W. Jacoby, M.D., of New York, and Robert T. Edes, M.D., of Washington.

The Association voted to become an integral part of the American Congress of Physicians and Surgeons, and Dr. Landon Carter Gray, of Brooklyn, was appointed delegate, with Dr. Charles K. Mills, of Philadelphia, as alternate, to attend the meeting of the

Conference Committee to be held in Washington in September next.

The Committee on Encephalic Nomenclature was continued, with the addition of Dr. E. C. Seguin, of New York, to fill the vacancy caused by the death of Dr. McBride.

The Association adjourned to meet at the call of the Council.





THE  
Journal  
OF  
Nervous and Mental Disease.

Original Articles.

CASE OF CHOLESTEATOMA OF FLOOR OF  
THIRD VENTRICLE AND OF THE  
INFUNDIBULUM.

BY WILLIAM OSLER, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA.

**C**LINICAL SUMMARY.—W. A. L., æt. 29. As a lad, had violent headaches which became more frequent about the eighteenth year. About this time several attacks of transient blindness. In 1876, '77 and '78, when a student, had trouble with his eyes, had headaches and would frequently fall asleep during the day. He graduated in 1879, and began practice. In June, sudden loss of power and sensation in left arm and leg, which returned in an hour. Shortly after, violent headache with vomiting. After an attack of somnolence, he had a brief maniacal outbreak. From July to October much headache, vomiting, and great drowsiness. Would sleep many hours. Pulse often as low as twenty-eight or thirty. Intervals of several days between the attacks. In October was at times incoherent, and lost all recollection of his wife and family. Sight much disturbed, and there was diplopia; one slight epileptiform convulsion. Throughout November and December great improvement, and rapid gain in flesh. For the first six months of 1880 he was able to be about, though the headaches recurred at inter-

vals, and the constant tendency to sleep persisted. Gait staggering. In March, double optic neuritis was determined by Dr. Buller. In beginning of July, another severe attack of headache and vomiting lasting three days, followed by a severe convulsion and prolonged sleep, from which he awoke quite blind. From this time rapid recovery of health, and for five years was able, though blind, to manage a drug business. On June 3d, 1885, return of attacks of headache, vomiting, and prolonged somnolence. Remained unconscious until August 27th, when he awoke at 4 A.M. quite suddenly. From this time pain in the head was the prominent symptom; no further loss of consciousness. Death suddenly, April 25th, 1886.

ANATOMICAL SUMMARY.—Cyst at base of brain in position of optic chiasm. Infundibulum greatly thickened. Small solid tumor in anterior and lower part of third ventricle. Dilatation of the lateral ventricles. Atrophy of optic nerves and tracts. Numerous pearly bodies scattered in the lining membrane of the cyst, and throughout the solid parts of the tumor.

The full account of this remarkable case is thus given by Dr. Buller of Montreal, and by the patient's brother, Dr. J. L.

The early history of the case, as related to me in a letter from the patient's brother, Dr. J. L., dated March 22d, 1880, is as follows: "My brother began to complain of his eyes about the beginning of the year 1877, and all the following summer he complained of more or less pain and uneasiness, but they did not give out until near the close of next winter. He was then in his primary year as a student of medicine, and found great difficulty in writing for his examination. His visual troubles continued to increase until about the month of May, when he went to Toronto to consult an ophthalmic surgeon, who pronounced his condition retinitis albuminurica. This diagnosis I never accepted, for it seemed unreasonable to me that he should have such advanced symptoms from a constitutional disease without having any of the physical or other symptoms of that affection.

“ Notwithstanding his imperfect vision he continued his course of studies, never missing a lecture up to the time he was first seen by Dr. Buller at Christmas time, 1878. His vision had then so far improved that he was able to read without difficulty. He completed his medical studies in the spring of 1879, having enjoyed excellent health the whole winter. Immediately after obtaining his degree, he commenced the practice of his profession in a country village, and all went well until about July 20th of the same year, when he was attacked with violent frontal headache and nausea with occasional vomiting; this continued for about a fortnight, accompanied by great lassitude and inability to make any exertion. It seemed an effort for him to exist. He had made up his mind to go to Ottawa for medical advice, and while waiting for the conveyance that was to have taken him there he fell asleep for a short time, and awoke in a violent attack of delirium, with complete suppression of urine, which lasted for about twenty-four hours. A brisk purge set him right, and the next day he left for Pembroke, where he arrived at my house the same night. It should here be mentioned that he had been vomiting his food for several days before his arrival here. This was about August 22d last (1879). The second night after his arrival, the pain became very severe, accompanied by uninterrupted vomiting for about eighteen hours. For several days he had repeated attacks of pain and vomiting, now always aggravated after a long and profound sleep. During the attacks of pain, the pulse would fall in frequency to about forty or forty-five, and even to twenty-eight and thirty; the temperature was not increased. (Probably any subnormal temperature would have been noticed by the writer of this letter if it had existed, but he does not speak of it.—F. B.)

“ In the intervals between the attacks of pain, his appetite would return, he would eat freely and apparently be improving. The first treatment he got some time during the first week here while suffering dreadfully. I applied six leeches and a blister, which gave immediate relief; in fact, the leeches had hardly taken when his pulse began

to rise, and in less than twelve hours he was perfectly easy.

"This condition continued with very little variation for about three weeks, when the attacks became milder and the intervals longer, but with a steady decrease of weight. I applied blisters repeatedly, always with marked benefit. I also applied a seton at the nape about the fourth week. On one occasion, about the fourth week, on attempting to stand up to pass water he was seized with a slight spasm, and I think if he had been kept in the upright position it would have developed into a convulsion, but immediately on his assuming the reclining position it passed off, which made me think it was from deranged circulation on account of his having suddenly assumed that position. About this time he complained of a loss of feeling passing all over his body; it used to alarm him very much, and he used to say, 'I cannot feel anything but my poor head.' It seemed to be a numbness lasting only for a little while; it occurred several times, sometimes all over the body, and sometimes only on one side. He never suffered from paralysis of any part or any organ with the exception of his sight. The special senses were all perfect the last time I saw him, about four weeks ago.

"About September 28th he began to show signs of mental failing, evidenced by slight loss of memory, and at times it was difficult to arouse him to perfect consciousness; he would mutter on being shaken, but you could not bring him to himself. This would continue for some hours, when he would wake up quite bright. This was his condition at intervals for the last week before I started to New York with him. The pain during this time was not very bad, and there was not much vomiting.

"He complained continually of feeling a sensation, in different parts of his body at different times, of the touch of what he called a pebble. He would describe the size of it as that of a pigeon's egg, with a rough surface. Of course he knew it was only an illusion, that it did not exist, but he had the sensation, and used to say he had a lump in his brain and that it was the size and shape of the pebble,

but the fact of his knowing the diagnosis was tumor would be a sufficient reason for him to connect the two and give rise to the idea.

"The morning of Oct. 3d, on preparing to dress him for the journey to New York, he was very poorly and could hardly realize that we were starting; indeed, before we left the house it was impossible to make him understand anything, and after we had started I decided to take the Perth train at Smith's Falls and return home, but before we arrived at Smith's Falls he wakened up quite bright and remained so until we arrived in New York on Saturday morning. He kept nicely all day Saturday and also on Sunday, which was the day we saw Dr. Janeway. He was then well enough to give the doctor a history of his case, but on Sunday night he began to suffer pain again, and for the next four or five days he vomited constantly and was at times more or less unconscious, still never so profoundly so as before leaving home, but he showed a dulness of perception of what was going on around him and partial loss of facts as to days, etc. I started for home Oct. 10th, and on moving him from one train to another I had to elevate his head and shake him. Once, in doing this, he had a distinct spasm; in fact, I think it might safely be called a convulsion. He remained more or less unconscious until we arrived at his own home in Perth on Oct. 11th. From this time until the 26th he was perfectly helpless in bed and quite unable to assist himself in any way. I was not sure if he was conscious when relieving himself; he apparently would recognize any person passing before his sight, but could not connect any ideas or think; took very little nourishment and was reduced to about eighty pounds—a perfect skeleton. Exactly a fortnight from the day we left New York, he opened his eyes on Sunday morning as bright as a dollar, and began from this time to eat, sleep, and gain strength. For eight weeks he gained flesh at the rate of one pound per diem until he weighed about 150 pounds. This change took place without treatment of any kind. After he began going about, he commenced taking iodide potass. up to

almost twenty grains twice daily, but not regularly, and another seton was put in the neck.

"From this time until Christmas he remained to all appearances perfectly well, without headache, nausea, or vomiting, and in the full enjoyment of all his faculties. About Christmas time he paid me another visit in Pembroke. After he had been here a few days, the headache and vomiting returned. He remained here a short time and then returned to Perth, where he remained until he visited Montreal."

I (Dr. Buller) saw W. L. for the first time about the end of December, 1878; he then appeared to be in good health and quite capable of carrying on his studies as a medical student. I was asked to examine his eyes in order to ascertain whether there remained any evidences of the retinitis albuminurica thought to have been discovered in the previous month of June. I could find no trace of disease of either retina or optic nerves. Vision was normal, refraction, H.  $\frac{1}{30}$ . I also examined the urine and found neither casts, albumin, nor sugar. The specimens examined under the microscope, however, contained numerous crystals of triple phosphates and large numbers of small octahedral crystals of oxalate of lime. At that time, he was not suffering from headache or any inconvenience from using the eyes for close work many hours daily. There was nothing in his manner or appearance to indicate a defective state of health. He next came under my notice on the 20th of March, 1880. His history during the intervening period has been given in detail in the foregoing communication from his brother. He came unattended to Montreal. The following day I noted his condition as follows:

The patient has a somewhat slow and hesitating manner of speaking; occasionally he forgets words that he should be familiar with, walks slowly, as if feeble and languid, and has a certain unsteadiness of gait which at times is almost staggering, especially on getting up after resting in a recumbent posture; at such times he feels a sort of giddiness. There is no evidence of weakness in executing

any ordinary muscular movements. The tendon reflex, however (knee jerk), is slow and weak. Complexion is rather fresh; the face has a puffy look and appears somewhat too fleshy for the body, and may best be described as a stolid heavy countenance entirely destitute of expression or animation; even when he smiles there is the same want of animation. At the same time, there is no defect in the voluntary movements of the facial muscles and no defect in cutaneous sensation. He still suffers a good deal from frontal headache, especially in the morning, and always carries the head somewhat thrown backwards. Vomited a little the morning he left home, but not since.

Four days later (March 25th) he was found to sleep most of the time, and when awake yawned very frequently. He is also much troubled with hiccough. The attendants in the hospital notice that he seems to forget to take his meals, and at times acts somewhat like a drunken man in his walk, and once or twice has almost fallen backwards when going up-stairs. In walking rather swings the legs. He attributes the uncertainty of gait to weakness—an idea that is perhaps not altogether without foundation, as the muscular power of hands tested with dynamometer only amounts to sixty pounds.

The appetite is fairly good, tongue a little furred, bowels inclined to be costive. Urine thirty-six ounces in twenty four hours, slightly acid, of a pale yellow color, deposits a little flocculent mucus, contains neither casts, albumin, nor sugar. There is no anomaly of sensation discoverable in any part of the body, and now he never feels "the lump" spoken of by his brother in the early stage of his complaint; is able to give a clear description of his past life; close questioning does not discover more than a possible venereal origin of the disease; it was, however, thought best to try the effect of iodide of potassium in full doses, commencing with twenty grains and increasing as rapidly as the stomach would bear the drug well diluted; this was commenced the second day after his arrival in Montreal. On March 26th he was examined by Dr. R. P. Howard, who gave me the following notes:

"Heart sound, normal; pulse, 65; presents no peculiarities; lungs healthy, but respiratory sounds weak; right side of chest flatter than left, and lower respiratory movements on this side markedly less excursive than on left side; shows an annoying restlessness under examination; has a papular(? iodide) rash on body and slight coryza; body emits a peculiar musty odor, which, however, is probably due to external circumstances. Is now taking iodide gr. xxx. thrice daily, preceded by a small dose of hydrocyanic acid a few moments before the iodide is administered. Still has hiccough and morning headache." The condition of the eyes was not placed on record until March 27th, but had not in any way changed since the 21st. It was as follows:

Pupils equal, in ordinary daylight about  $2\frac{1}{2}$  mm. wide, act sluggishly both to light and acc.,  $V = \frac{2}{3} \frac{0}{0}$  and  $Hm = \frac{1}{2} \frac{1}{8}$  each. The ophthalmoscope shows well-marked double optic neuritis—choked discs—not neuroretinitis, the swelling being little wider than the normal disc and quite steep. With hyperopia =  $\frac{1}{2} \frac{1}{8}$  at macula, the surface of the nerves is best seen with + 10. Veins dark and tortuous, but of normal size; arteries a little smaller than normal; vessels only here and there hidden or obscured by the swelling of papilla; no hemorrhages, and only a moderate degree of white striation, and the papilla appears rather reddened; macula regions entirely normal. There is no contraction of the visual fields, no defect in perception of colors, and the muscular system of the eyes presents no abnormality.

He remained in Montreal until April 3d without any material change in his condition; some days feeling a little better and others suffering more from headache (always frontal), occasionally vomiting, was taking pot. iod. gr. lx. three times daily, when he returned to his home in Perth.

Oct. 21st. Came to Montreal again for the day in order to have another examination of the eyes, having now become entirely blind. Continued taking the iodide in about the same doses all summer, but for the last three weeks has omitted it. Vision failed steadily from the time he



left Montreal, but could still see fairly well about the beginning of July, when he had another severe attack of headache and vomiting which lasted some three days, and culminated in a convulsion. This was followed by a profound sleep from which he awoke entirely blind, which has continued up to the present time. Since this last severe attack his general health has steadily improved. Has had no headache to speak of since the end of August, only a little occasionally just on the top of the head; feels strong and well; walks without staggering; his countenance has gained in expression; is well nourished, and in the matter of appetite and sleep there is nothing amiss; also avers that sexual power is unimpaired. The appearance of the optic nerves has undergone a great change: both are alike extremely pale, scarcely if at all swollen, a little irregular at the margin. The veins tortuous, but both veins and arteries much diminished in size. Was next seen by me on June 24th, 1882; came on account of an acute catarrhal otitis media of the left ear, which has caused him a considerable degree of pain for the past ten days, otherwise his health has been very good since his last visit. The completely atrophic optic nerves have never afforded him a glimmer of light since the day he became blind. The ear trouble yielded readily to the usual treatment, and he returned home on June 24th.

The remainder of the history is thus given by his brother, Dr. J. L. "He recovered perfectly from the ear trouble and remained well, enjoying good health until June, 1885—making five years of relief from his trouble—when the pain reappeared, and up to the 27th of August, he suffered much as in the first illness, with severe attacks of pain, vomiting, and long spells of somnolence. During some of these attacks the pulse was very weak and fluttering, and in one it was thought that he was going to die, and I was telegraphed for. He was more or less unconscious all this time, and it is said that when the attack passed off on the 27th, the first word which he spoke was to take up the sentence he left off in June, three months before, when seized with the headache. On the 28th of August he sat

up and took his dinner at the table, and remained well, with the exception of slight attacks of pain until Nov. 15th, when he was seized with a terrible stabbing, piercing, unendurable pain in the head and his face flushed crimson. This gradually passed off, and he was able to walk to the post-office. From this time he was up and down, one day well and several days in pain, but there was very little vomiting and no disturbance of the pulse. He slept well when not suffering.

"During the last month the intervals of relief were very short, a day or two at most, and the attacks of pain longer, and for the last fortnight the pain was nearly constant; he has to have some person sitting beside him to keep him from falling asleep; if he happened to fall asleep, in a few minutes he would wake up *frantic* with the increased suffering. The Thursday before he died he was down-stairs enjoyed his meals, and he looked quite well, and likely to be so; he was always so cheerful and bright when free of pain and suffering. He passed away without any struggling or any particular warning of the approach of his death. He had peculiar attacks, the last three weeks before his death. I was at his bedside one morning, and he called me in distress and complained of a strange feeling in his head. He said his head was all drawn up, and that his face was also all drawn up, although showing no indication or appearance of any change in expression. His hands and feet were extended and rigid, but could be flexed by force. He appeared greatly alarmed and distressed, and his appeals of distress and alarm were pitiful. During the attack, which lasted half an hour, his pulse never varied or changed; it was perfectly normal. The attack lasted about half an hour; it returned again several times during the day. Another expression he used, 'My inside is all drawn up.' He used the word *drawn* to describe the sensation in his inside, face, and head. He had several attacks of this character the last two weeks before his death, and it was fearing an attack like this, and feeling it coming on, that he called his mother the night of his death: he said,

"Mother, mother, I am going to have one of those attacks; raise me up!" He then said, 'I feel like fainting, get me a glass of water!' He tried to drink it, but it came back, his head fell forward and he passed away, and never moved again."

Dr. Fraser, of Perth, Ont., has also written an interesting account of the patient's last illness which practically corresponds with the above description.

I happened to be in Montreal the day on which Dr. L. received word of his brother's death, and as I had seen the case on several occasions with Dr. Buller, I gladly consented to go to Perth with Drs. Buller and Wyatt Johnston to make an examination.

*Autopsy*, twenty hours after death. Body well nourished. Face and general surface blanched, rigor mortis present, *calvaria* of normal thickness. *Dura mater* not very adherent. Sinuses contain fluid blood. Surface of brain as examined *in situ*, symmetrical, but rather wide in parietal regions. A large quantity of clear fluid escaped in removal of the organ. A few adhesions of the pia mater and brain substance to the dura covering the middle fossæ so that the brain substance here tore in lifting out the temporo-sphenoidal lobes. No adhesions at the base, but the infundibulum was greatly thickened, and cut with resistance at its point of junction with the pituitary body.

Parts of the base present the following appearance: Olfactory bulbs look small and the nerves seem a little flattened, particularly the posterior third. A cystic tumor, the size of a walnut, occupies the space between the corpora albicantia and the commencement of the longitudinal fissure. It measured about three by three centimetres, and consisted of two parts, an anterior cyst, somewhat translucent, and a posterior firmer, cone-shaped portion which represents the infundibulum and was attached to the pituitary body by a stalk five millimetres in thickness. The mass occupies the position of the chiasma, no trace of which can be seen. The optic nerves are atrophied, only two millimetres in diameter, gray in color, and were at-

tached to the antero-external angles of the cystic tumor. The optic tracts pass off from the postero-external portion and as far as the anterior fibres of the crura are distinct, but from this point they are represented by a thin, pale, gray bands, scarcely discernible. In front the tumor presents a rounded smooth surface, which rests upon the longitudinal fissure, and the hinder part of the first frontal convolutions. Laterally it does not extend upon the anterior perforated spaces. Posteriorly it reaches the corpora albicantia, but does not involve them. The crura form part of its posterior boundary, and they look as if slightly spread by it. The pia mater covers the mass, but is not specially adherent or thickened. The vessels of the circle are a little displaced, but are otherwise normal. The nerves at the base appear healthy; the left third nerve looks a little translucent at one spot.

The convolutions are slightly flattened, and the vessels of the pia not unusually full. On section the substance cut with firmness. Centrum ovale looks natural, puncta vasculosa numerous. Corpus callosum normal. Lateral ventricles are considerably dilated, and contain an excess of fluid. The posterior cornua seem particularly large. The veins along the surface are full. Fornix and septum are flattened, but can be lifted readily. Velum interpositum very vascular, and the venæ Galeni full. The third ventricle presents the following condition: Pineal gland, with its peduncles, and the posterior commissure look normal. The middle commissure is large and distinct. A firm mass occupies the anterior and lower part of the ventricle between the pillars of the fornix. It is about 2.5 centimetres in length by 1.5 in breadth. Behind it is in contact with the thalami, and on the right side with the smooth surface of the caudate nucleus. The right pillar of the fornix is distinct, the left appears to be involved, and the mass is of greater extent towards this side where it is firmly connected with the caudate nucleus. It is solid in the greater part of its extent, but centrally there is a cyst with clear fluid. Whether this

originally communicated directly with the third ventricle could not be determined, but at the upper part the wall is very thin and translucent. The cyst is directly continuous with the one at the base of the brain.

The corpora striata and optic thalami appear normal.

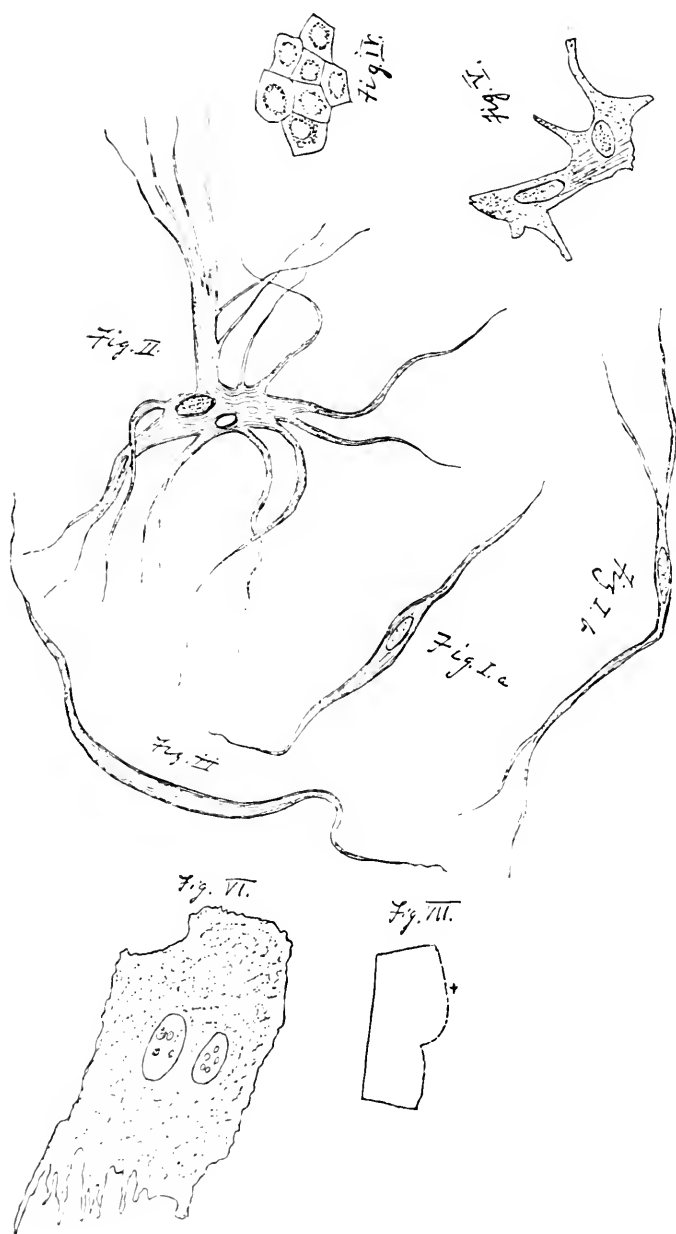
The tumor then occupied the anterior extremity of the third ventricle, partially involving the left pillar of the fornix, but not extending laterally into the ganglia. At the base, it involved the parts known as the tuber cinereum, the lamina cinerea, and the infundibulum, destroying completely the chiasma and producing wasting of the optic nerves and tracts.

The infundibulum forms a firm conical mass attached to the hinder part of the tumor, and tapers to a diameter of five millimetres at its insertion into the pituitary body.

The tumor consists of a cyst with solid walls of variable thickness and clear contents. Whether it communicated with the third ventricle was unfortunately not accurately noted, probably it did not. The lining membrane of the cyst is smooth and glistening like that of a ventricle, and here and there in the surface are small yellow granules.

At the base, anterior to the thickened infundibulum, the walls are very thin and translucent, but laterally and behind they form a firm, solid mass of a grayish color and present a rough, uneven surface. The portion in the third ventricle has thicker walls in proportion to the cyst, and the tissue has a grayish translucent aspect. Through the wall in places, particularly where thin, a yellow color is noticeable, not uniform, but in small areas. The tissue of the infundibulum is solid, gray externally, but yellowish-brown in the inside and on section it cuts with a gritty sensation.

Pons normal. Fourth ventricle and corpora quadrigemina present nothing special. The *iter* not much dilated. The posterior aspect of the cord, about twelve millimetres below the calamus scriptorius, presents a very remarkable depression, as if a fine tight cord had been



passed round in an oblique direction, extending from a point just above the line of emergence of the anterior roots of the first cervical nerve. The part above the constriction projects seven millimetres beyond the level of the rest of the cervical cord. The pia mater dips into the depression, and the outlines of the funiculi graciles and restiform bodies are quite distinct to its margins. There is no softening, no hyperæmia, no alteration in color, and it looks like an anomaly rather than a pathological condition. Fig. 7 shows a facsimile outline sketch after section in the groove between the restiform bodies and the posterior column on the right side.

*Histological examination.*—The tumor consisted chiefly of: (1) a matrix of densely interwoven fine fibres without definite arrangement. In the infundibulum and on the wall of the cyst they were more closely set than in the softer mass within the third ventricle.

(2) Spindle and branched cells which were found in all parts, but more particularly in the softer portions by the base of the cyst and in the ventricle. From the latter situation, teased bits showed very remarkable forms; many were fusiform, greatly elongated and with the extremities prolonged into delicate filaments (Fig. 1, *a* and *b*). Some of the branched forms were the largest and most beautiful structures of the kind which I have ever met with in either normal or pathological growths. Fig. 2 represents one of these large "spider" cells outlined with the camera. Many of the processes were prolonged far beyond the margins of the field. The protoplasm was as a rule delicate, with but few granules. Here and there were noted curious elongated non-nucleated cells with a hyaline, homogeneous stroma (Fig. 3). I have described these as occurring in a case of medullary neuroma of the brain,<sup>1</sup> and have since met with them in several gliomas.<sup>2</sup> They probably result from the transformation of the ordinary spindle cell, many of which are identical in form.

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<sup>1</sup> Journal of Anatomy and Physiology, London, vol. xv.

<sup>2</sup> Medical News, Phila., 1886.

(3) A beautiful pavement epithelium (Fig. 4) lined the cyst; the cells were not extremely flattened, and in many places were filled with granules.

(4) Pearly bodies which were attached on the inner wall of the cyst, and were also very abundant in the thickened infundibulum. These consisted of nests of epithelial cells, and as many of them were calcified, section with the knife gave a gritty sensation. The concentric arrangement was well seen in the smaller nests, but not in the larger ones, which were too deeply impregnated with lime salts. The epithelial elements were very numerous in the thickened infundibulum, and all shapes and sizes occurred in teased preparations. Many were much flattened and curved; others of irregular and bizarre form (Fig. 5). Some of these were of comparatively enormous size and very flat (Fig. 6). It was difficult at first to believe that we were dealing with epithelial cells. It is interesting to note that there were no cholesterin crystals. The remarkable indentation in the posterior aspect of the upper part of the cord, an outline of which is given at Fig. 7, showed in section a normal white matter at the base of the groove without a trace of induration or increase in the fibrous elements.

*Remarks.*—Indications of brain trouble existed in this case for at least ten years, and possibly the headaches which occurred when a lad may have been due to the growth in the third ventricle either beginning or assuming a more active condition. During the year 1879 and the first six months of 1880, the growth extended to the base of the brain, and produced at first neuritis and finally atrophy of the optic nerves. This was due to the gradual formation of the cyst which occupied the position of the chiasma. At this time, too, the headache was most intense, the signs of irritation (convulsions, paræsthesia, vomiting, staggering gait) most marked. Recurring attacks of somnolence occur with great frequency in brain tumor—particularly in syphiloma, but I do not think we have yet reached a satisfactory explanation of their variability. We may reasonably assume that from July, 1880,



to June, 1885, the brain accommodated itself to the increased pressure, and that during this time the growth remained stationary. The return of the symptoms in 1885 may have been connected with the development of the hydrocephalus due to pressure of the tumor on the veins. A portion of the mass in the third ventricle looked recent, and certainly contained less of the dense fibrillar connective tissue than in other parts, indicating possibly a more recent formation. I thought at first that the constricted furrow on the upper portion of the cervical cord might be due to pressure, and in this way might perhaps explain some of the symptoms of tingling, etc., of which he complained; but the situation and character of the groove and the absence of the slightest induration are very much opposed to such a view.

The *tumeur perlée* of Cruveilhier, or cholesteatoma of Johannes Müller, is a very rare growth, most often met with at the base of the brain. It is in reality an endothelioma, and in this instance probably began in the cellular lining of the third ventricle, and its extension in the infundibulum.

#### EXPLANATION OF THE FIGURES.

FIG. 1, *a* and *b*.—Spindle cells from the mass in third ventricle.

FIG. 2.—Enormous "spider" cell from the same situation. Nos. 7 and 3.

FIG. 3.—Non-nucleated, translucent fibre cell.

FIG. 4.—Endothelial lining of the cyst wall.

FIG. 5.—Irregular form of endothelium obtained by teasing a small piece of the central part of infundibulum.

FIG. 6.—Enormous flat endothelial scale. Nos. 9 and 3.

FIG. 7.—Outline of medulla and cord showing the furrow in the posterior surface; + indicates the *calamus scriptorius*.

# ON THE IMPORTANCE OF THE CORPUS STRIATUM AND THE BASAL FORE-BRAIN BUNDLE, AND ON A BASAL OPTIC-NERVE ROOT.<sup>1</sup>

BY DR. L. EDINGER,

FRANKFORT-ON-THE-MAIN.

**M**EYNERT was the first to apply the comparative-anatomical method to the study of the course of nerve tracts in the brain. From the very start, this method brought to light many interesting facts of fundamental importance; and yet, strange to say, very few investigators have employed this method. At the present time, we may claim that the work done during the last decades has advanced our knowledge of the gross anatomy of the brain to such a point as to enable us to determine the homologous parts of the cerebral organism throughout the entire animal series. There can be no doubt now which parts of the brain represent the mid-brain, which the inter-brain, and such difficulties as remained have been finally removed by the clever researches of Rabl-Rückhardt. These difficulties were greatest concerning the interpretation to be given to the various portions of the brain of osseous fishes; but even here we can now see clearly. In a former publication<sup>2</sup> I have shown why it is that in spite of a considerable number of investigations our knowledge of the course of nerve tracts in the animal brain is so much more limited than that of the external configuration of the brain. It was shown that even in the lower animals, the Selachians for

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<sup>1</sup> This paper was read by the author at the twelfth annual meeting of the S W. German Neurologists, etc., at Strassburg. Translated by the Editor.

<sup>2</sup> Anat. Anzeiger, 1887.

instance, the structures lying caudad of the fore-brain were so complicated that their relations were scarcely simpler than those of analogous portions of the brain in man and mammals. My own researches, with Weigert's staining methods, upon the lower animals, including a study of frogs, amphibia, and reptiles, have forced me to the conclusion that the structure which subserves the simple so-called lower functions of the nervous system is substantially the same in all animals. Little headway can be made in these studies even with the lower animals if adult individuals are examined, as any one can convince himself who will take the trouble to study the rather complicated section through the mid-brain of Selachians. If one adopts, however, the method of comparative embryology and investigates the development of medullary sheaths in lower animals, the relations of the parts exhibit unusual simplicity and clearness. In this way I have succeeded in unravelling a very considerable portion of the central nerve tracts of the sensory nerves of the brain.

It is my object in the present paper to discuss another question in cerebral anatomy which we can solve more satisfactorily still by the aid of comparative anatomy.

It is well known that there is considerable controversy regarding the fact whether nerve fibres actually emanate from the corpus striatum of the fore-brain, or whether the corpus striatum is merely a way station through which nerve fibres pass. Up to the present time, the solution of this question has been attempted by the examination of the brains of adult or growing mammals. The great difficulty that we have to contend with in such investigations is that we have to take into account the innumerable fibres of the corona radiata, the greater part of which fibres unquestionably merely passes through this ganglion. That this is true of a very large portion of the fibres coming from the cortex I was able to establish by the study of foetal human brains.<sup>1</sup> In the seventh and eighth months of pregnancy, the cerebral structure is

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<sup>1</sup> Neurol. Centralbl., 1884, No. 15.

still a very simple one: but later on, so many different sets of fibres acquire their medullary sheaths that the successful staining of these brains reveals a complicated network which it is scarcely possible to unravel. In spite of the fact that certain researches, and particularly the embryological relations of the corpus striatum, argue in favor of the view that fibres issue from the putamen and from the caudate nucleus, as they do from the cortex, yet on account of the difficulties above mentioned it was quite impossible to determine with absolute certainty which view was the more correct one.

The cerebral structure is very much simplified among some of the lower vertebrates. Rabl-Rückhardt showed

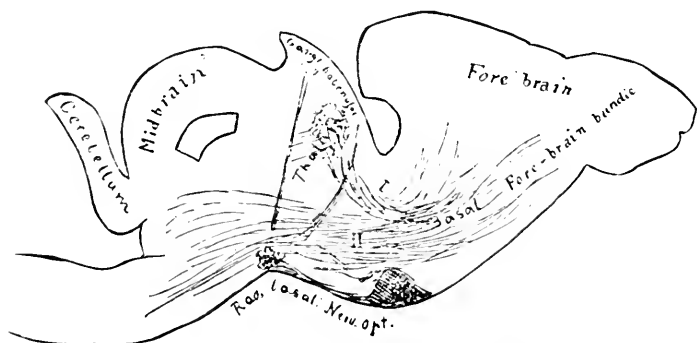


Fig. 1.—Brain of *Emys lutaria*, sagittal section considerably laterad of the median line.

some years ago that osseous fishes possessed no cerebral mantle with nerve fibres, and my own researches on amphibia and reptiles have taught me that the mantle of these animals contains but very few medullated nerve fibres. In these various animals the main mass of the fore-brain is made up in reality only of the corpus striatum which has a hemispherical nucleus. In reptiles, starting from this nucleus rows of ganglion cells grow into the cerebral mantle. These animals are, therefore, lacking in those very features which complicate the mammalian brain: they are devoid of a corona radiata extending inward from the cerebral mantle.

There is no difficulty in showing in amphibia, reptiles,

and fishes that a *well-marked bundle of fibres does actually issue from the corpus striatum*. The course of this "basal fore-brain bundle" could be traced most easily in reptiles (*Lacerta*, *Anguis*, *Emys*, *Tropidonotus*). From the prosencephalic ganglion the basal fore-brain bundle passes caudad. In the inter-brain it divides into a coarse-fibred portion (I. in all figures) which ends in the ganglion of the thalamus, thus uniting thalamus and lenticular nucleus, and into a bundle of finer fibres (II. in all figures) which can be traced about as far downward as the oblongata, but in all probability extends even further down.

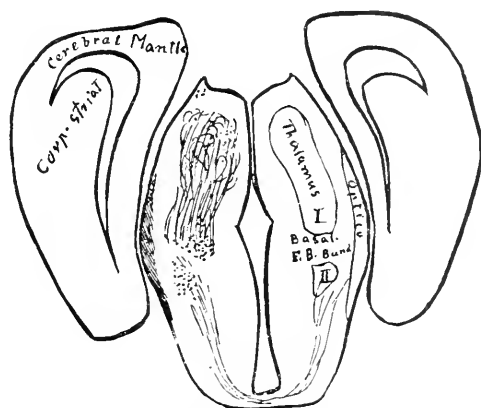


Fig. 2.—*Emys lutaria*, frontal section through the inter-brain and the posterior portion of the hemispheres.

Immediately before their entrance into the thalamic ganglion a few very fine fibres are found between the right and the left bundle, which fibres present the appearance of a transverse commissure; but I am not convinced that this commissure, which has been seen only by Osborn before me, arises from these bundles. This commissure lies on the floor of the inter-brain, dorsad and frontad of the commissure of Gudden which is immediately adjoining the chiasm. If we designate the commissura gangliorum habenulæ as the dorsal commissure of the thalamus, and call the commissure of Gudden the ventral commissure of the thalamus, then we might give to the commissure described above the name of "median commissure of the thalamus" (commissura thalami medialis). This commissure is not to be confounded with the commissura *mollis* common to reptiles. The commissura thalami medialis I have found in all animals which I have thus far examined (fishes, amphibia, reptiles, and birds). It is particularly well

marked in Selachians ; its homologue in mammals I have not yet made out.

The following conclusions can safely be drawn from these investigations which have been made on simple transparent specimens :

1. Fibres originate in the corpus striatum.
2. The corpus striatum is doubtlessly joined to the thalamus.
3. A bundle of fibres can be traced from the corpus striatum caudad nearly as far as the oblongata.

The conditions above described as occurring in verte-

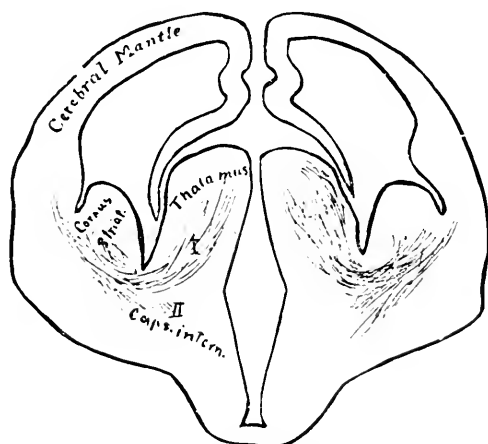


Fig. 3.— Human fœtus of two and a half months. Frontal section through the inter-brain and the hemispheres.

brates are present also in man. Meynert long ago drew this inference, basing it upon specimens of adult brains; but, as was said before, innumerable fibres are there crowded together into a very small space so that nothing can be stated with any degree of certainty. The accompanying frontal section of a human fœtus of two and a half months exhibits very distinctly that of the fibres emanating mainly from the corpus striatum the greater portion passes into the thalamus, whereas the remaining fibres pass lower down through the internal capsule.

It is interesting to observe that not only in man and in

mammals, but also throughout the entire animal series these fibres from the fore-brain ganglion become medullated at a very late period, later than the fibres, for instance, from the inter-brain and mid-brain. Among the amphibia, a very considerable portion of these fibres never becomes medullated (*Salamandra*, *Bufo*).

If we are anxious to get at the physiological function of this bundle of fibres, it would seem to be important to examine animals that lead an active independent life during their foetal period. It is a very surprising fact, therefore, that not a single trace of medullary sheaths can be discovered around the fibres of this basal fore-brain bundle in the larvæ of the frog, salamander, triton, slow-worm (the latter even thirty days after their birth), and ammocœtes; and furthermore that the axolotle exhibits only very few medullated nerve fibres among an enormous number of medullated fibres.<sup>1</sup> All these animals lead an independent existence; they swim, creep, have perceptive and visual powers, take nourishment, escape, flee from the approaching hand; in short, they perform a number of actions in spite of the fact that their fore-brain is joined in very imperfect fashion to the remaining portion of the cerebrum.

The embryological development of the human fore-brain proves that medullary sheaths are developed whenever certain portions of the brain come into active use. If this be correct, the larvæ spoken of above represent highly organized animals that do not make use of their fore-brain.

Of late years, the cerebral physiology of lower vertebrates has aroused considerable interest. It would be desirable if more attention were paid in future, than hitherto, to the inner anatomical structure of the brains here under discussion. In this way only will it be possible to determine the function of this fore-brain bundle

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<sup>1</sup> In the new-born sparrow and in the pigeon that has just emerged from the egg, this bundle is still without medullary sheath. In the amphibia, triton, and bufo, the greater portion of this basal fore-brain bundle remains non-medullated during the entire period of life.

which, since it is present in all animals, must be of fundamental importance.

After reading the above paper and in connection with a communication made by Stilling on the origin of the optic nerve, a report was made on a well-marked optic-nerve root in reptiles: the same root I have seen in amphibia and fishes. It arises at the base of the brain from a ganglion which must be interpreted to be the corpus mammillare, since it lies between the tuber cinereum and the emerging oculo-motor nerve. This ganglion is connected by a distinct bundle of fibres with the ganglion habenulæ, from which the optic nerve for the parietal eye of reptiles arises. This can be made out in lizards and tortoises. Fig. 1 exhibits this basal optic-nerve root.



## ILLUSTRATIONS OF ERROR IN THE DIAGNOSIS OF SOME NERVOUS DISEASES.

BY IRVING C. ROSSE, M.D.,

WASHINGTON, D. C.

A FEW years since, a distinguished neurologist of New York, Dr. William A. Hammond, called my attention to the frequency with which chronic inflammatory and degenerative affections of the spinal cord are incorrectly diagnosticated and treated as rheumatism.

This fact, made more impressive by seeing and examining at the Post-Graduate Medical School a number of patients who had been treated for rheumatism by practitioners otherwise well informed, has been of great diagnostic importance to me in my neurological career, and I purpose in this paper to reinforce Dr. Hammond's assertion, by presenting a few observations of my own which have been selected during the last two years from a large number of cases in which there was notable failure in the diagnosis.

The cases in question were examined for medico-legal reasons, and each was accompanied by more or less voluminous written testimony of physicians who had recorded such diagnoses as chronic rheumatism, rheumatism and heart disease, rheumatism and disease of eyes, malarial poisoning, disease of the liver and spleen, sunstroke and resulting loss of sight, general prostration and debility, sciatica, and other vague pathological generalities. These certificates of disability were written by medical men from widely distant points of the United States and by surgeons of the army and navy.

For convenience I have divided the cases into three

groups; the first including those in which the lesion is confined to the posterior columns, the second those in which there was a more general spread of the sclerosis, and the third those in which cerebral symptoms were a prominent feature.

## I.

CASE I.—A man of 44 came under notice on May 11th, 1885. For a number of years he had been ineffectually treated for "rheumatism and resulting disease of the heart." He complained of pains in the chest, with shortness of breath and pains in all the joints; of a "dead-like" feeling and no use of limbs; of lightning-like pains in legs, tenderness in back, and a feeling as if a ramrod were in his spine.

The objective symptoms showed no external changes from rheumatism, and the patient appeared to be fairly well nourished. The pulse, from excitement of the examination, was accelerated (112), and the heart's action somewhat irregular; respiration 18; temperature normal. The area of the cardiac dulness was extended transversely, and a musical bruit accompanied the period of both sounds of the heart over the region of the mitral valves. There was apparent tenderness over the lumbar spine, a tabetic walk that became more aggravated when the eyes were shut; and an absence of the patellar tendon reflexes. Various tests showed loss of sense of the topographical relations of different parts of the body and corroborated so plainly the lesion of the æsthesodetic system that a tyro ought not to have failed to recognize the existence of a tabetic affection.

CASE II.—In the next case of supposed "rheumatism," seen June 4th, 1885, the development of locomotor ataxy was more rapid. It was that of a man of 39 who was taken sick January 16th, 1885, and paid but little attention to his limbs till March, when he had to use crutches. He complained of continued pain in both upper and lower limbs and in the back of neck, and of sharp darting pain in the lower extremities; of pains and palpitation of

heart, shortness of breath, loss of appetite and sleeplessness; of impaired eyesight and of inability to walk since March 1st of the current year.

At the time of examination, the patient was confined to bed and in an acute febrile condition. His temperature was 102.2°, pulse 112, respiration 24; tongue coated.

There was tenderness on pressure over first dorsal vertebra; some atrophy of muscles of the lower extremities; complete absence of the patellar tendon reflexes, and an inability to touch tip of ear or nose with the eyes closed. Parallelism of the optic axes perfect; no paresis of ocular muscles, and the eye grounds were normal in appearance. Vision equal to  $\frac{19}{xx}$ . Complained of a slight halo around candle, but vision seemed not to be seriously impaired. There was, however, well-marked myosis, the pupils being almost unaffected either by darkness or light. This iridoplegic sign, taken in connection with the history of the case and the presence of other tabetic symptoms, left but little doubt as to the diagnosis.

CASE III.—The next case, seen Sept. 15th, 1885, is that of a man of 58 who had also been treated for “rheumatism.” He complained of resting badly at night and of being scarcely able to turn in bed; the left arm and leg were worse than the right. Said that he sometimes falls down, and that his whole body is sensitive, but that he has no swelling of the joints.

Objective manifestations supported many of the patient's statements. There was nothing abnormal about either respiration or temperature; but he was much emaciated, and motor inco-ordination was shown by a painfully tabetic walk and inability to stand without invoking the supplementary aid of the eyes. There was loss of knowledge of the topographical relation of parts of the body, with apparent tenderness over lumbar spine; inability to differentiate æsthesiometric points, and an entire absence of the knee-jerk. I was unable to find any structural change indicative of rheumatism.

CASE IV.—On October 5th, 1885, another instance of the kind in a man of 44 came under my notice. This man

had been treated five years for "rheumatism and disease of the eyes," which disabilities he alleges were contracted in Colorado. He complained of shooting pains in the legs, of great difficulty in walking, and of double vision.

Examination showed loss of sensation in the lower extremities, inability to stand or walk with eyes shut, absence of knee-jerk, and a tottering, unsteady gait. The action of the heart was forcible and increased, with a musical systolic bruit over the apex, and the area of cardiac dulness was increased; no external evidence whatever of rheumatism. Eye tests showed O. D.  $V = \frac{15}{xx}$ , O. S.  $V = \frac{10}{xx}$ . There was homonymous diplopia from inco-ordination of oculo-motor nerves, also some atrophy of both optic nerves.

CASE V.—On October 29th, 1885, a man of 28 was the next tabetic patient seen. He had a medical record of "rheumatism, impaired eyesight, and general debility." He stated that he had become sick in China, and that, on account of his staggering gait, he was often accused of being drunk, though perfectly sober. He complained of sharp pains extending from the small of back to feet; of sharp, tingling pains in the soles of feet, and of a sensation of tightness around waist. Gives out easily. Eyesight is much impaired. Has occasional spells of vomiting, and throat is occasionally troublesome. Besides these symptoms, is troubled with a painful discharge from hæmorrhoids. Admits having had a soft chancre, but never any constitutional symptoms.

In this case, no evidence of rheumatism was found. The tabic walk betrayed the patient before he had begun his tale, and examination revealed an inability either to stand or walk, or touch tip of nose or ear with index finger when the eyes were closed. Knee-jerk absent. Heart's action excited, but no valvular lesion. Right eye showed vision equal to  $\frac{15}{60}$ , when myopia is corrected; the left,  $\frac{15}{45}$ , when myopia of  $\frac{1}{16}$  is corrected. Both optic disks blanched and indicating well-advanced atrophy, particularly the right. Structure of both eyes myopic. A large crescent on margin of right disk, and a smaller one on that of left.

Another visit from this man, on May 27th, 1886, showed

an aggravation of the symptoms. He had undergone some emaciation, being 5 feet 10 inches and weighing but 129 pounds. Complained of constant pain all over body, but mostly in legs. It is occasionally sharp and darting, like needles sticking in legs. Is dizzy and unable to walk without a cane or other support. Toes feel as if too big for shoes.

Further examination only strengthened the previous diagnosis. The tendon phenomena, motor inco-ordination, and slight disturbance of the sensibility were more marked than previously. The heart's action was excited and irregular, with a systolic bruit over its apex. Vision of left eye  $\frac{1}{90}$ ; right,  $\frac{3}{150}$ . Both myopic about  $\frac{1}{18}$  for left. With this correction,  $V = \frac{1}{48}$  for left. Neither eye further improved by any lens. Nasal side of both visual fields much impaired; temporal side of visual field somewhat contracted. Color sense fair. Large crescent on margin of each optic papilla, and the disks, particularly the right, were blanched, indicating well-advanced atrophy. No change in region of macula lutea. Media clear; pupils movable. No cicatrices on glans penis or on groins, but the cervical and inguinal glands and those of the epitrochlear space were engorged.

CASE VI.—My next "rheumatic" case is that of a man of 45, who was seen on January 14th, 1886. He also was much under weight, being 5 feet  $9\frac{3}{4}$  inches and weighing but 120. For some time, his lower extremities, according to his statement, had been partly paralyzed, and he was unable to use himself, being obliged to have help when he goes out. Cannot stand to wash his face, because shutting of eyes, in doing so, makes him tumble over. Is losing his memory.

The ataxic and other symptoms of tabes dorsalis were too evident in this case to bear recital.

I saw the patient again on January 28th, when he complained of weakness and numbness in his legs; of gradually diminishing eyesight; of his fingers seeming all thumbs when he tries to pick up anything; of the same inability to

wash his face without sitting down; and of being very sensitive to cold.

At this juncture, there was noticeable, in addition to the other tendon phenomena, an absence of the cremasteric reflex. The left foot had become everted. No tenderness on pressure over spine; but impaired sensation of skin. No evidence of syphilis. Vision of right eye,  $\frac{20}{xx}$ ; left,  $\frac{2}{xx}$ . Refractive media clear. Optic nerve of left eye much blanched, and calibre of retinal vessels much contracted, indicating advanced atrophy of optic nerve. Atrophy of right eye not so advanced. No evidence of choroiditis nor retinitis. Decay of vision result of tabes dorsalis. Evident mental impairment.

CASE VII.—The next case, that of an old stager, a former captain in the army, who had been the rounds of many physicians, was seen January 20th, 1886. His army medical record showed him to have suffered from "malarial poisoning, rheumatism, disease of heart, disease of the eyes, also bladder or urinary organs, and of liver and spleen," for all of which he drew an invalid-pension from the government. His history, when compressed, amounted simply to this: pains in eyes and legs and in urinary organs; sometimes sees things double.

All his symptoms showed well-advanced dorsal tabes. No splenic or hepatic enlargement. No external evidence of rheumatism. Is fairly well nourished; 5 feet 9 inches; 157. Action of heart excited and irregular; a systolic bruit over its apex; pulse 120. Incontinence of urine. Atrophy of right os calcis.

CASE VIII.—Incontinence of urine was also present in a case seen February 16th, which for many years had been treated as one of "malarial poisoning." The patient stated that he has to urinate frequently during the night; has cramps in feet and toes; has been impotent for last six months, and is losing his memory. Is sensitive to cold; vomits at times, and throat troubles him. ad partial paralysis of left side about eighteen months ago, and about three months since an attack of erysipelas.

This man was 45 years old, 5 ft. 5½ inches high, and

weighed but 96 pounds. His tabetic walk was difficult and unsteady; the usual defects of co-ordination and of diminished sensibility were present, as well as the existence of marked melancholia. The eye-grounds were, however, normal in appearance and there was no loss of co-ordination in the ocular muscles. Hearing of right ear  $\frac{1}{4}$  s; left  $\frac{6}{4}$  s. Both tympanic membranes normal in position and appearance; heard conversation at 20 feet. No splenic or prostatic enlargement and no hepatic symptoms. Incontinence of urine and this excretion loaded with phosphates. Catheter No. 9 passes readily into bladder. No evidence of syphilis.

CASE IX.—This case, under date of February 18th, 1886, is that of a corpulent army officer aged 59, whose record shows "heart disease resulting from injury of left side." He said that his whole left side is weak and lame; is always in pain; does not sleep well, is uneasy, cannot lie on left side, and has shortness of breath.

Examination failed to reveal any traumatic evidence of the alleged cause of the patient's condition. The pulse was 78, and the heart's action regular, but increased on exertion. Cardiac sounds normal, no increase in area of dullness, but evident irritability. There were present the ataxic symptoms consequent upon degeneration of the posterior columns of the spinal cord; atrophy of muscles of the left side of the body, more particularly in the gluteal region, and partial ptosis of left upper eyelid.

CASE X.—Seen March 3d, 1886. An emaciated and anæmic subject, whose former disabilities are reported to be "rheumatism, disease of the heart and lungs, incontinence of urine, and partial loss of sight." His testimony is that he has pain in the knees and stumbles on attempting to walk, pain in chest, and cannot sleep on either side. Pain extends around waist, has shortness of breath and spits blood. His urine dribbles away constantly, eye-sight is bad, being unable to see at night.

The patient's condition manifested itself by the characteristic tabetic stagger on attempting to walk with closed eyes, absence of knee-jerk, tenderness over lumbar spine,

involvement of vesical sphincter, and disturbed sensibility. Vision of right eye  $\frac{1}{20}$ , left  $\frac{1}{15}$ . Hypermetropia  $\frac{1}{4}$ . With this correction vision of right eye equal to  $\frac{1}{20}$ , left  $\frac{1}{15}$ . Optic nerve of left blanched, with evidence of some atrophy. Considerable loss of visual acuity in right eye, but its ground not indicative of marked trophic change in nerve. Area of cardiac dulness much increased; apex beat transmitted to border of ensiform cartilage; musical systolic bruit over apex. Valvular lesion with hypertrophy. No dulness, but rather an exaggerated resonance over both lungs, with distinct mucous râles. No external evidence of rheumatism. Case speaks for itself.

CASE XI.—Another instance of "chronic rheumatism and resulting diseases of the heart" in a fairly well-nourished man of 51 came to me April 3d, 1886. He complained of severe pain in head and shoulders and across the small of back, and of severe vertigo. Heart almost jumped out of mouth at times when excited, and he has shortness of breath all the time.

The objective facts of this case failed to support the allegation of rheumatism, no external evidence whatever being present. The area of cardiac dulness was somewhat increased, action of heart excited and irregular, some roughness over region of mitral valves. Sensory and motor symptoms those of locomotor ataxy. Hypermetropia of both eyes equal to  $\frac{1}{2}$ . With this correction, however,  $V = \frac{1}{20}$  and cannot be improved further by glasses. No trophic change in optic nerves, although some engorgement of retinal veins amounting to simple hyperæmia, a condition of vision probably owing to cerebro-spinal lesion.

CASE XII.—In the following case, seen June 28th, 1886, the patient, a medium sized man of 59, was thought to have incurred "affection of the eyes and disease of the heart and lungs from a gunshot wound of the head," received in the late civil war. His principal complaint was difficult breathing on walking and almost total loss of sight. No history of syphilis.

On stripping for examination, a small ventral hernia, an inch in diameter, was noticed three inches above the um-



bilicus. Area of cardiac dulness considerably increased, apex beat being felt below ensiform cartilage. Distinct bruit over apex indicating lesion of mitral valves. Dulness over apex of left lung, with bronchial breathing and slight mucous râles. Right lung more resonant, but vesicular murmur only faintly audible. Imperfect expansion of chest and some apparent tenderness on percussion over both lungs. There was a non-adherent V-shaped cicatrix on the scalp just over the craniometric point known as the *lambda*, and to this wound the patient attributed his ill health. Ocular examination showed vision for right eye to be  $\frac{15}{180}$ , that of left  $\frac{15}{180}$ , and both eyes slightly improved in vision by a lens of plus  $\frac{1}{2}$ . There was marked atrophy of both optic disks, particularly of the left, the papilla being blanched and the calibre of the vessels much diminished. Color sense impaired. Field of vision fairly good, but somewhat contracted on nasal side. The Argyll-Robertson symptom was present in a marked degree, with absence of the knee-jerk. The impaired sight in this case, when considered in relation to the other symptoms observed, can hardly be attributed to the effects of the gunshot wound.

CASE XIII.—A blind man of 60, whom I saw August 19th, 1886, stated that he had been unable to see for nearly twenty years, and that the only treatment he had had was for "sore eyes," which pained him continually, and that he had also had pain in his head, temples, and back.

Patient was much emaciated, being six feet and weighing but one hundred and five pounds; pulse 90; respiration somewhat quickened; temperature normal. No evidence of syphilis. Vision for both eyes 0. Complete atrophy of both optic papillæ. Disk whitened, and retinal vessels shrunk to mere capillaries. Has convergent strabismus of left eye. The disturbance of sensation, loss of thermic sensibility, disturbance of muscular sense, and the presence of symptoms common to tabetic patients, leave but little doubt as to the diagnosis.

CASE XIV.—In this case, one of "sciatica and piles," which came under observation on Feb. 7th, 1887, the

patient's venereal history was not so good. He was a man of 45, and said that sciatica does not trouble so much now as it did; but he had great difficulty in walking, with shooting pains in his limbs at times, and that in the dark he goes all to pieces.

The symptoms in this case were displayed in a manifestable way. There was the tabetic walk, the knee phenomenon, and inability to stand erect or walk, or to direct the movements of the limbs with precision when the eyes were shut. Vision for both eyes  $\frac{20}{20}$ , and these organs were normal excepting the pupils, which were abnormally small (myosis). Glands of epitrochlear space enlarged; also other evidence of syphilis.

CASE XV.—The antepenultimate observation of this series concerns a retired naval officer, aged 51, who was seen by me on May 31st, 1887. A medical friend tells me that he has known of this case for the last twenty years, and that the patient has been treated for "partial paralysis, the result of injury to back and resulting disease of the heart."

Briefly told, the patient's symptoms were pain in the back, head, and limbs; inability to control feet and legs in walking; and difficulty in retaining his urine.

Tabic symptoms were here shown by the characteristic walk, the tendon phenomena, and by increased muscular inco-ordination on closing the eyes. Patient stout, but anæmic and feeble, and tongue tremulous when protruded. Pulse, sitting, 84; standing, 120, and heart's action so forcible after slight exercise that its pulsation could scarcely be counted. A musical bruit over its apex which was displaced downwards and to right of normal position. Also increase in area of cardiac dulness. Vision for both eyes  $\frac{12}{20}$ . Has H. for both  $\frac{1}{2}4$ , with which correction V is improved to  $\frac{20}{20}$ . In other regards, eyes are normal. Mind seems sluggish; beyond this, intellectual functions apparently intact.

CASE XVI.—The last recorded instance of the kind that I shall mention came to notice a few weeks since, on June 3d. It is that of a physiological bankrupt of 63 years, the

diagnosis of whose complaint appears to have been a matter of extreme difficulty, since a mere enumeration of the various ailments for which he had been treated would take a large slice from the nosological table. Some of his disabilities were "deafness, disease of the left side the result of malarial fever, and resulting affections of the head, heart, and left knee."

The salient points of this patient's malady, as described by himself, were a whirring noise in both ears; pains in the whole left side of body, including the left side of the head, at which point it was not severe; extreme palpitation of heart after exertion; and almost total inability to breathe at times, when he feels like swooning away.

Cardiac and gastric disturbances were well marked in this case, with debility, insomnia, and tremulous tongue; difficulty and unsteadiness in walking; tendon phenomena; muscular inco-ordination; and the usual symptoms that go to show the existence of locomotor ataxy.

## II.

The cases mentioned in the following group are not typical records of lesion in the kinesodic system; but the symptoms, both subjective and objective, seem to indicate the presence of combined sclerosis in which the spread of the disease to the antero-lateral columns had complicated the lesions of the posterior columns. In the majority of these cases, both patient and physician mistook the disability for rheumatism.

CASE I.—"Weakness of the back and stoppage of urine" were the diagnostic interpretations that caused a man of 40 to consult me on June 6th, 1885. He had been ailing for about ten years, and complained principally of loss of power in his legs, which appeared worse at night. The trouble in urinating not so great as formerly.

There was tenderness over dorsal spine; difficulty in co-ordinating muscular movements without invoking the supplementary aid of the eyes; muscular tremor, and exaggeration of the patellar tendon reflexes.

CASE II.—A man of 59, and fairly nourished, came to

me Dec. 3d, 1885, having been treated several years for "rheumatism." He complained of pains in his spine, chest, legs, and knees, and of shooting pains around waist and passing down into legs; of shortness of breath, and of great difficulty in climbing a stairway.

Careful examination failed to seize upon any symptoms that would justify a belief in the presence of rheumatism. The action of the heart was, however, excited and irregular. Area of cardiac dulness increased; a musical systolic murmur over its apex, with obstructive bruit over aortic valves. Other symptoms, those characteristic of spinal sclerosis.

CASE III.—Another of alleged "chronic rheumatism." March 16th, 1886. Man 49 years. Has pains in back and right side, particularly in right leg. Unable to walk without crutches. Sensation of band around waist.

No evidence of rheumatism. Area of cardiac dulness increased. Loud systolic bruit over apex of heart, action of which is excited, irregular, and much increased on exertion. Extreme shakiness and muscular motility; inability to control muscular movements unassisted by his eyesight; impaired cutaneous sensibility in lower extremities, and ankle-clonus in left leg.

CASE IV.—A large, well-nourished man of 51 was brought to me in a completely helpless condition on April 21st, 1886. He stated that he had for a long time been treated for "rheumatism," and that for the last two years he had been paralyzed from the small of his back downwards. No pain at present; but had formerly and very severe at times; sleeps well, appetite good, bowels constipated, but both bladder and rectum emptied without difficulty.

On examination, the lower extremities appeared normal, although the paraplegia was such as to prevent either walking or standing, the paralysis both of motion and sensation being nearly complete. Tenderness over lower portion of spine. Heart normal.

Under the use of the actual cautery applied to the spine and a course of static electricity, this patient is almost

entirely recovered at the present writing (July 6th). He complains of slight numbness only in the soles of his feet, and of slight muscular inco-ordination. He, however, stands and walks well with his eyes closed, and the patellar tendon reflexes are normal.

CASE V.—On May 20th, 1885, I was consulted by a medical man who lived in Mexico, and had been treated for “nervous prostration and diabetes.” He stated that he is also subject to diarrhœa alternating with constipation, that he has pains in the small of back, frequent micturition, numbness of the extremities, palpitation of the heart, and loss of vision.

His symptoms showed nothing abnormal in temperature, pulse, or respiration, he was well nourished and 55 years of age. Tongue slightly furred, abdomen protuberant, but neither tender nor tympanitic. No splenic or hepatic enlargement, no piles. Action of heart irregular and forcible, musical murmur over apex with its first sound. Vision for both eyes  $\frac{9}{1250}$ . This great loss of acuity owing to high degree of hypermetropia  $\frac{1}{6}$ , correction of which increases V to  $\frac{15}{50}$ , and to pannus involving both corneas to some extent, the latter disease being the result of severe granular conjunctivitis which has converted conjunctiva of both lids and balls into cicatricial tissue, causing also partial symblepharon and some xerophthalmia. Urinary analysis showed sp. g. 1.018, albumen 0, sugar 0, color sherry, and reaction acid. There was an inability to open and shut eyes alternately; exaggerated knee-jerks, and other symptoms characteristic of spinal sclerosis.

CASE VI.—November 15th, 1886. Still another case of “rheumatism” in a man of 37, who complains of a dead feeling all over, and of trouble in his head and eyes, the latter becoming gradually worse.

Morbid emotional disturbance in this case was very marked, the patient having sobbed on finishing his statement. Esthesiometric examination showed impairment of the cutaneous sensibility in the lower extremities. Patellar tendon reflexes exaggerated. Slight general mus-

cular tremor. No evidence of rheumatism or of syphilis.

Patient was seen again on April 4th, 1887, when he still complained of a dead throbbing feeling in legs and over heart, and of pains in head. He stated that he gets easily tired and cannot talk without his tongue getting all tangled up.

As at the previous examination, the heart was found to be healthy, and there was no evidence either of rheumatism or syphilis. The pupils were more dilated than normal, but no contraction of visual field, vision being  $\frac{20}{xx}$  for both eyes. In addition to former symptoms, marked disturbance of speech was noted, the disease having impeded the innervation of the lips and tongue to such an extent as to produce the peculiar defect of articulation sometimes observed in such cases.

CASE VII.—February 12th, 1887. Another case of "chronic rheumatism," but this time the previous diagnosis was not quite on the *lucus-à-non-lucendo* principle, since some traces of articular rheumatism were apparent. The patient, aged 51, complained of pain all through his body; of inability to see with his right eye, and of being all crippled up.

Examination showed a somewhat corpulent person with a pulse of 120, which increased to 150 on slight exercise. The joints of the left hand were enlarged; the right could neither be flexed nor extended, and the grasp of the left was much impaired. Right arm atrophied one inch and a quarter, and temperature of right forearm somewhat lowered. Heart's action forcible and excited, with slight roughness over the apex, but no hypertrophy or pronounced valvular disease. There was great exaltation of both superficial and deep reflexes, with ankle-clonus, intention tremor, and muscular inco-ordination. Vision of right eye 0; that of left  $\frac{1}{3}\frac{5}{10}$ . The latter improved to  $\frac{1}{2}\frac{6}{10}$  with  $+\frac{1}{2}\frac{1}{4}$ . Right optic disk blanched, the vessels so shrunk in calibre that arteries are scarcely perceptible. Cornea has an opacity over pupillary area. Left optic disk somewhat blanched, indicating incipient atrophy. Optic axes diverge; projection very bad.

CASE VIII.—This case, hitherto recognized as “rheumatism,” was in the person of a broken-down old man of 71, with arcus senilis and paralysis agitans. He called on me March 18th, 1887, complaining of pain and stiffness in all his joints; of piles and dizziness, and of inability to exert himself without great pain in the back.

Inspection failed to reveal any external evidence of rheumatism. Action of heart forcible and intermittent, with musical bruit over its apex; increase in area of cardiac dulness and transmission of apex beat to ensiform cartilage. No psychic or sensory symptoms; but absence of knee-jerk and presence of muscular inco-ordination.

CASE IX.—The following case, in which paralysis agitans had been incorrectly diagnosticated, came under notice May 4th, 1887. It was in a large man of 57, who stated that his tremor is so great as to prevent his either reading or writing; and that in addition thereto he is troubled with a sense of constriction about his heart, and with shortness of breath.

Objective signs showed both hypertrophy and valvular lesion of the heart. Entire body from head to foot was in a tremulous condition, the head being uncontrollable except when held. In brief, the symptoms present were rather those of disseminated sclerosis.

CASE X.—If I may be allowed to express myself in the form of an hibernicism, I should say that the last case of “rheumatism” mentioned in this group is one of progressive muscular atrophy.

It occurred in an ill-conditioned man of 53, whose subjective symptoms were pain in the eyes and neck, nervous prostration, deafness, and troubles of eyesight, being at times unable to see at all, and at others seeing double.

Cardiac hypertrophy with valvular disease was present; but there were no external indications of rheumatism, and patient declared that he does not now suffer from that cause.

There was general muscular tremor with fibrillary twitchings, and the atrophy was most marked in the muscles of the right leg. Vision of right eye  $\frac{8}{120}$ ; left  $\frac{12}{20}$ .

Projection bad; divergence of optic axes; pupils sluggish. Well advanced atrophy of both disks, and diminished calibre of retinal vessels. Hearing, both ears  $\frac{0}{48}$ . Both tympanic membranes opaque and sunken, with narrowing of both Eustachian tubes.

### III.

The cases of the concluding group, in which cerebro-spinal symptoms predominate, were all incorrectly diagnosed; rheumatism, malarial poisoning, and sunstroke being the principal factors of error.

CASE I.—A man of 38 complained to me on May 19th, 1885, of the results of a horse bite on the right forearm, which he had sustained about a year previously in Kansas. Among his symptoms he mentioned constant darting pain in right arm and leg; a feeling of a band around his waist; incontinence of urine; and of incessant motion of the right arm and leg, which kept up even during sleep.

Observation showed the scar on the forearm to be of a trivial nature. There were complete right hemiplegia; general muscular inco-ordination; exaggerated reflexes; spinal tenderness, and a spasmodic action of the affected side, which may have been post-hemiplegic chorea, but bore a stronger resemblance to athetosis.

CASE II.—The next case seen by me, June 2d, 1885, was that of an Englishman of 23, who a year previously, in one of the Western States, had suffered from "apoplexy and paralytic stroke." He was unable to speak; but he indicated by signs that he had pains and tremors in his right side.

He was fresh colored, apparently well nourished, and there was nothing abnormal about pulse, temperature, or respiration. Muscles firm, limbs symmetrical, and no appreciable difference in temperature of either side. Walks with difficulty. Writes with right hand and uses it with tolerable facility in dressing and undressing. Intellect seemingly not much impaired, and though aphasic, he makes audible but inarticulate replies to questions.



I saw this man again February 18th, 1886, at which time both the aphasia and agraphia had increased; he was unable to express himself either verbally or by writing except to a limited degree. Amimia was not yet very marked, as he could communicate by gesture to some extent, and gave me to understand that he can no longer write with the right hand, that he is deaf in right ear, and cannot see well with the eye of that side.

He replied by writing with the left hand short but barely intelligible answers to several questions, and did not speak beyond saying "No." Is still well nourished; no diminution of muscular volume in right half of body, which is hemiplegic. Vaso-motor disturbance and mental impairment also noted. Since patient could make no statement, it was difficult to ascertain with certainty the condition of his sight and hearing. The vision for left eye appeared to be 1; that of right  $\frac{1}{15}$ . Hypermetropic astigmatism of the latter, but no positive evidence of atrophy of optic nerve. Hearing, right ear  $\frac{0}{48}$ . Tympanic membrane normal in position and color. Left ear, hearing  $\frac{30}{48}$ , and membrane same as in right. Deafness probably owing to lesion of portio mollis.

A third examination, September 29th, 1886, showed patient's condition in every way worsened. He was unable to communicate his condition except by imperfect signs; from one hundred and fifty his weight had decreased to one hundred and thirty pounds, and enfeeblement of the intellect was more marked.<sup>1</sup> The otoscopic signs were those previously noted. Ophthalmological examination showed impaired vision with incipient atrophy of the optic disk.

CASE III.—On September 2d, 1886, another instance of aphasia with mental impairment came under my notice. It was that of an ill-conditioned man of 40 years, whose disability had been reported as "sunstroke and resulting loss of sight of eyes, and general prostration," for which he had been discharged from the army.

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<sup>1</sup> This man having since been accused of a criminal offence, a question arises as to his responsibility.

The subjective symptoms were still more difficult to get in this case than in the preceding one, owing to the patient's "general prostration," which consisted in utter helplessness and apparent complete aphasia, agraphia, and amimia.

There was nothing abnormal in pulse, respiration, or temperature, excepting coldness of the extremities; tongue much furred; sensation of touch and of pain greatly diminished; can raise hands to mouth and has considerable grasp; knee-jerk exaggerated; incontinence of urine; right oculo-motor nerve paralyzed with divergent strabismus of right eye and ptosis; optic axis of right eye diverted outwards and downwards; eyes practically movable in this position; disk somewhat blanched, that of left eye normal. He has vision in each eye as proved by his grasping objects presented to him; but it is impossible to determine the exact amount of loss, owing to the patient's abnormal cerebral condition, which prevented expression and so clouded his intelligence that it was with difficulty that he could be aroused.

CASE IV.—In the next case (September 17th, 1885), there was something more than "rheumatism and disease of the heart and eyes." The patient, a slight man of 34, with nothing abnormal as to pulse or respiration, complained of being unable to get around without assistance, owing to the uselessness of his right arm and leg, and pains in the back caused by every jar of his foot. He also alleged shortness of breath on slight exertion, constipation, incontinence of urine, spinal tenderness, and poor eyesight, which was particularly so in right eye.

Examination failed to reveal any symptom of rheumatism or heart disease. There were, however, spinal tenderness, loss of motion and of sensation in both upper and lower extremities, with vaso-motor disturbance and diminution of temperature, atrophy of the hemiplegic muscles, dyskinesia, exaggerated knee-jerk, and ankle-clonus. Vision of right eye,  $\frac{1}{10}$ ; left,  $\frac{1}{3}$ . Engorgement of right papilla amounts almost to choked disk, its margin being cloudy and scarcely definable. Left optic papilla

showed less vascular disturbance, but disk itself somewhat blanched and indicative of slight atrophy.

CASE V.—“Malarial poisoning, rheumatism, and resulting disease of heart” perhaps never showed themselves with more vagueness than in the following case:

April 14th, 1886. Man of 54; occupation seaman; hails from a malarious locality in Virginia; is 5 feet  $8\frac{3}{4}$  inches high, weighs 150 pounds. Says he has lost all use of right side; that his heart thumps; is short-winded; sleeps badly, and has lost his memory.

This man's complexion was somewhat sallow and his tongue slightly furred; but there were no splenic or hepatic symptoms, nor any hæmorrhoids, evidence of rheumatism or of heart disease. Muscles of right side considerably atrophied; circumference of leg two inches, of arm one inch less than that of left. Sensation of right half of body impaired. Well-marked ataxic symptoms, exaggerated knee-jerk, and ankle-clonus. Hearing right ear  $\frac{6}{48}$ ; left  $\frac{6}{48}$ . Both tympanic membranes much sunken, and handles of mallei thrown into sharp relief. Left Eustachian tube much narrowed. Presence of nasopharyngeal catarrh accounts for impaired hearing. Evident mental impairment, disorder of the memory being very marked.

These cases speak for themselves. It will, however, be seen that many details concerning previous condition, such as the patient's family history, atavistic antecedents, alcoholic and nicotinic habits, and the like, have been omitted. This was purposely done, not only for the sake of brevity, but more particularly because of the irrelevance of asking questions that might have suggested symptom pictures to a class of patients whose interest it was to magnify their ailments.

I have dwelt at some length upon the condition of the eyes in the cases under consideration; for it seems to me that a study of the alterations of the eye, as shown by means of the perimetric records of the visual field, and by the ophthalmological images of the intraocular changes

found at its posterior segment, has done so much to advance our knowledge of nervous diseases that the signs developed by this mode of inspection are almost pathognomonic, and should for that reason take a high rank in diagnosis. Of course, it is not pretended that the intra-ocular changes by themselves are of absolute significance any more than a subcrepitant râle or a bronchial inspiration, yet they lend to the diagnosis of cerebro-spinal disease an additional source of correctness, and, to say the least, are important complements to other symptoms. Taken collectively and associated with other symptoms furnished by the patient, the lesion of the nerve structure of the eye confirms the diagnosis by corresponding presumably to what is taking place in other parts of the nervous system. In a recent article on "cerebroscopy"<sup>1</sup> I have endeavored to show the importance of studying the alterations of the eye in connection with nervous diseases.

Iridoplegia and the condition of the eye-grounds, taken in connection with the knee phenomenon, may in certain instances give rise to confusion, notably in the case of negroes, among whom exists a certain obtusion of peripheral sensibility probably corresponding to the flattening of the tactile corpuscle, which renders it difficult to disturb the negro organism by a reflex action. Two persons of this race suffering from locomotor ataxy having lately come under my notice showed absence of the knee-jerk with other tabic characteristics; but on subsequent examination of fifty-two other negroes who had no symptom of nervous disease, to my surprise, I succeeded in getting the knee-jerk in but one instance, and that only after interlocking the fingers and causing traction.

Cerebral and mental symptoms are noticeable features in a considerable number of the cases herewith mentioned. In Case VI. of the first group dizziness was a subjective symptom, while vertigo played a similar part in Case XI. Amnesia and hebetude were found in Cases VI. and VIII.

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<sup>1</sup> "Reference Handbook of the Medical Sciences."

of group one; mental impairment with disorder of the memory in Case V. of group three; aphasia with mental impairment in Cases II. and III. of group three; morbid emotional disturbances in Case VII. of group two, and melancholia in Case VIII. of the first group.

These mental symptoms, taken in connection with the others—sensory, motor, and trophic—herewith recorded, may be interpreted as medical facts that affect criminal responsibility and questions of civil incapacity, and if they have no other significance they at least show in the matter of chronic affections of the spinal cord that, so far from being “one of the most thoroughly understood in the whole range of medical science,” and their recognition “one of the easiest problems of neurological differentiation,” as we are told by most recent and trustworthy authority, they are, as a matter of fact, questions the solution of which appears to have been one of difficulty and error among practitioners both far and near.

1732 H ST., N. W.

## PAINFUL INSANE IMPRESSIONS DUE TO MORPHIA.<sup>1</sup>

SAMUEL B. LYON, M.D.,

BLOOMINGDALE ASYLUM.

I WILL present this evening some notes in a case which interested me especially because of the consistency of the delusions and terrors with the apparent immediate cause, and because of its sudden and complete recovery.

Mrs. Blank was 50 years of age. She had for three years suffered from repeated and most severe attacks of pain about the heart, which had been diagnosed as *angina pectoris*, and for which all manner of remedies had been used with no great advantage, except from morphia. As morphia gave relief, she was advised to use it freely to control the pain, being informed that recovery from her disease was not probable, and that any means of making the remainder of her life comfortable was justifiable. Under this advice, I was told, she took each day from two to seven grains, divided into three doses, and in addition she inhaled, at times, as much as six ounces of chloroform in a day. This treatment for her attacks of pain was continued for nearly three years before her mind began to be affected.

Her mental trouble began with hallucinations of both sight and hearing, and these were of such a nature as to produce delusions of extreme terror. Her hallucinations and consequent delusions related at first to her family, but later embraced all her friends and acquaintances. Her own language best describes her state of mind, and I will quote it. "I began to believe that every one connected with me—at first my children, later my more distant

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<sup>1</sup> Read before the New York Neurological Society, October 4th, 1887.

relatives, and still later my friends and acquaintances—were compelled to suffer because they were connected with me. I have seen my son encased in an iron armor which I thought was filled with spikes, which contracted upon him, and I would hear him turning a crank which caused its contraction, and I could hear him going up the street in this armor, which rattled as he walked. I saw him chained in the yard, and thought he lived on the stones he broke; and yet all the time I knew and remembered, and do now everything which occurred about me. I thought my son was a prisoner and condemned to be shot, and I heard the shots fired and voices say, 'Charlie is dead,' and I saw his body brought back. This is an example of the torture I saw inflicted upon all my children, but the variety of torture varied with each one: I thought my married daughter lived in a tree; that she had lost her mind; that her baby had been taken from her, and it was also made to endure some awful suffering." Day after day new sufferings were added to those the patient endured, and finally a clear and well-defined scheme of torture, directed as much against her feelings as against her body, was evolved and put in practice. She was informed that she herself was first to undergo great bodily suffering in the presence of her daughters, in order that they might suffer in seeing her so tortured, and that her own agony should be intensified by the observation of theirs. New instruments and wild animals were daily added to the collection of means of carrying out this cruel purpose, and these were always in her view, and the nature and purpose of each was told to her and discussed before her. Her imagination later added a new terror to its already rich collection. She grew to believe at length that every one who spoke to her, who was kind to her, or whom she even looked at, or thought of, became as a consequence involved in the dire calamity which was to overwhelm at first herself, and later her family and friends. I will mention very briefly some of her other painful ideas, which to her had all, and more than all, of the intense reality which ordinary perceptions have to well minds. She said that she saw

a cross to which she was to be screwed through innumerable parts of her body, a new screw being added each day. A cobra was to be wound around her body. Every one she thought of was to be tortured, and the effort not to think of persons, only brought them into her mind, and caused their ruin, and she would then hear herself reproached by these innocent persons for their misfortunes.

She appears at this time to have had a double or two-sided consciousness. She took an interest in and was fully aware of all that went on about her, of the persons she saw, and the general tenor of the affairs of her family; and that she was particularly observant was shown later, after she had come to the hospital, where I knew her. Here, in new surroundings and among entirely new persons, she very soon learned much about them, their family relations, etc., and the general routine of the place. As an instance of this, I will mention that, as she explained afterwards, she tried to avoid meeting the writer, because she thought she would not only involve him in the impending ruin, but would also cause the destruction of his wife whom she had never seen, but of whom she had learned, and who was to suffer simply because the patient thought of her. With this clear cognizance of the ordinary events and persons about her, she, in the other side of her consciousness, lived in a realm of fantasies, peopled with all the horrors of the damned.

These two states of mind seem to have been balanced against each other, with the weight on the side of the unreal state, as long as she continued in her own house among the chief objects of her solicitude, and under the influence of the drug to which she appears to have owed her misery.

At first the scenes she witnessed were simple perceptions, each standing disconnectedly, but later she reasoned out a motive, an agent, and a system of action, the appreciation of which scheme intensified beyond measure her agonies. There was an acquaintance of the family, a man whom she had, when well, always disliked, because she



believed him to be of a cruel nature, his treatment of animals and various small actions of his having given her the ground for this dislike. He had in reality never done her or hers any injury, but now, in her belief, he became the principal actor and director in the machinations against her, in which he was unremitting and without mercy.

When Mrs. Blank was received at the hospital, she was a most characteristic case of melancholy, her eyes were fixed upon the floor and rarely raised, her head was bent, her voice was low and only heard in reply to direct questions, her general condition was delicate but not feeble. Physical examination revealed no organic disease. While in the hospital she was removed from the probably provoking cause of her mental trouble, viz., the morphia, and the contributing causes, viz., the presence and anxious solicitude of her family, which fostered the intense self-consciousness of the patient—a quality as prominent in the most pronounced melancholia as in the most exalted megalomania or paresis—and soon a gradual improvement took place in her condition; her appetite and sleep improved, and her expression of countenance showed obviously less despondency from day to day. She became more willing to talk, and to listen to the repeated assurances that her fears were groundless. At length, one day, she seemed to banish the whole thing, her face brightened, and she was ready to discuss her former feelings and beliefs, having an intense relief that they were gone, but still the nervousness of one who has passed through a great danger safely. At that time she gave the details of her former state of mind, and her language was noted by me as of much interest.

It is curious perhaps that, while insane, she had no such attacks of pain in the region of her heart as she had experienced before her insanity developed, and that a few days later, and after all mental symptoms had disappeared, she had an attack of this pain after having taken a little hurried exercise, which pain did not yield to the internal remedies used, but did to an application of the electrodes of a small medical battery placed over her heart. Her imagination,

as much as the current, having probably afforded the relief. Some subsequent paroxysms of this pain have taken place, but, notwithstanding these, she appears to-day as well and contented as any rather delicate lady would ordinarily appear.

The particular points in her case which I have thought interesting are: 1st. The likeness of her hallucinations and delusions to those described as being produced at times by opium, but which, with her, persisted through weeks instead of for a brief time, as is usual. Toxic insanity has been said by a recognized authority to be in no way distinguishable from insanity from other causes. This case is the exception which proves the rule, the symptoms bearing a direct relation to the cause, viz., the particular poison.

2d. Her almost instant shaking off in a day of her morbid ideas, as she explained, upon the assurance of the writer that they were not to be credited; but it is to be remembered that the improvement in her general condition had prepared the rational side of her mind to make this last effort successfully.

3d. Whether a genuine case of angina pectoris, dependent on disease of coronary arteries of the heart, would alternate with a distinctly nervous condition.

4th. Whether the unlimited use of morphia, or any other poison, to alleviate pain is to be recommended.

## TWO CASES OF BRAIN TUMOR.

(1) TUMOR OF THE SECOND FRONTAL GYRE; (2) TUMOR OF THE OPTIC THALAMUS. REMARKS ON THE LOCALIZATION OF OCULO-MOTOR AND FACIAL CENTRES.

By CHARLES K. MILLS, M.D.,

NEUROLOGIST TO THE PHILADELPHIA HOSPITAL, ETC.

THE specimens herewith presented are of unusual interest from several points of view. If the symptoms exhibited by the patient from whom the first specimen was taken were due to the only lesion which I was able to find, the case is unique, as it shows oculomotor paresis and upper facial paralysis apparently due to a cortical lesion, or to a lesion conjointly of the frontal cortex and centrum ovale. The patient from whom the second specimen was removed had also marked paralysis in the upper distribution of the facial nerve as well as the lower, and this was associated with hemiplegia and some hemianæsthesia of the same side.

CASE I.—*Tumor of the Second Frontal Gyrus.* W. V., æt. 16 years, male, was admitted to the Philadelphia Hospital about two weeks before his death. His mother died of heart disease; his father is living, and is a hard drinker. He had two sisters and one brother, who are healthy; another brother, however, died in infancy of hydrocephalus. He had measles and scarlet fever about eight years ago, and dropsy followed the latter. He had been compelled to work beyond his strength for years. He had had occasional convulsions for over a year, and thought that he had received a blow on the head before they came on. He had a scar about an inch long in the scalp on the right side

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<sup>1</sup> Read before the Pathological Society of Philadelphia.

of the head, but it seemed to be superficial; no depression or cleavage of bone could be made out.

On or about December 27th, 1886,<sup>1</sup> in trying to rise from his chair, he fell. He had noises in his head, or, as he expressed it, "his head moaned." He also had vomiting at this time, and obstinate constipation; and sound and light were intensified by the pain in his head. He had some fever, and his pulse was sometimes as low as fifty to the minute. The next day, there was some loss of power, and he walked with a staggering gait; he had headache and cold sweats. On the third day, he had diplopia, vertigo, and palpitation; and he now cried out with severe pain in his head, and could retain nothing on his stomach. He also had seizures, the nature of which could not be determined. These got somewhat better, but were followed by sensations of pins and needles in the arms, accompanied with great stiffness. His bowels continued to be much constipated. In the second week of his illness, he lost power, to some extent, in his left arm, and had some depression of sensation in the right. Although during this week he seemed to be stronger, on attempting to walk he staggered and fell. He had intolerance of light all the time, especially marked in the right eye. During his fits, he was drawn to the left side, and always cried out before they came on. He had sensations of choking during the fits, and said that he was never unconscious in them. He was ravenously hungry most of the time during his illness after the first week, and bolted his food at all times.

His temperature on admission was 99°, and afterward ranged between 98° and 99°. He had attacks of vomiting at irregular periods, and his bowels were much constipated, but readily moved by enemata. His eyes were somewhat immobile, making his gaze seem fixed. His pupils were equal. He had a slight but distinct ptosis of the right eye, sometimes more marked than at other times. He could not close the left eye, nor wrinkle the left forehead. He

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<sup>1</sup> During the early part of his illness, he was attended by Dr. E. H. Speer, of Philadelphia, who kindly sent me some notes of his case.

had distinct loss of power in the left arm and hand, and also in the left leg, but not so marked. He died apparently from general exhaustion.

*Autopsy.*—The body was that of an emaciated boy; there were several scars on the head. Slight adhesions of the pleura were found over the upper portion of the right lung; the lungs were otherwise normal. The heart, kidneys, and liver were normal. The intestines and spleen were not examined.

The dura mater was not adherent to the skull, but, on attempting to remove it, the right side was found to be strongly adherent to the pia mater and the brain underneath it over the antero-frontal region, and the corresponding portion of the median surface of the right hemisphere. In removing the brain, a little serous or grumous fluid exuded from this region, but no true pus. On carefully removing the dura, a reddish-gray grumous mass was found adherent to the agglutinated dura and pia mater, and involving the gray matter of the brain and a portion of the white matter immediately adjacent. This was soft, and in its centre a hole or cyst appeared, or the space had been occupied by tissue now broken down. The exact location and dimensions of this mass were as follows: It was almost entirely confined to the posterior portion of the second frontal gyre (of Ecker), encroaching very slightly at its posterior and lower part upon the lower third of the ascending frontal gyre. Its greatest dimension was, diagonally from below upward and forward, three and a half inches. Perpendicularly, the broken-down mass extended two and a half inches, and also two and a half inches antero-posteriorly. It involved the white matter of the second frontal gyre, and to some extent that of the first and adjacent portion of the third. The ganglia and capsules proper were not involved. There were some meningeal congestion and inflammation continuous from the mass.

The existence of ptosis of one side, and of paralysis of the upper fibres of the facial nerve of the other, with paresis of the arm and leg of this side, led me to infer that

the case was one of more or less diffused meningitis, with exudation of the base. It looked as if the facial and oculomotor nerves of opposite sides, at their superficial origins, had been more or less involved in some process causing pressure to be exerted upon them, and that the descending motor tracts were so affected as to produce paresis of arm and leg. The most careful scrutiny of the base, examining one nerve after another at its origin, and as far as possible along its course, revealed nothing, and it is reasonable to infer that the symptoms present were due to the only lesion discovered, namely, the tumor involving the posterior part of the second frontal gyre, a small portion of the ascending frontal gyre, and the adjacent white matter. It is not my purpose to go into any lengthy comments upon the case, but rather to submit it as a clinico-pathological contribution to the study of localization, to be used by others or myself at some future time. It is unfortunate that no ophthalmoscopic examination was made in this case. Arrangements were made to examine the eyes carefully with the ophthalmoscope, but, through a combination of unfortunate circumstances, it was not done.

According to Ferrier,<sup>1</sup> at the base of the first frontal and extending partly into the second frontal gyre in the brain of the monkey is an area, irritation of which causes elevation of the eyelids, dilatation of the pupils, conjugate deviation of the eyes, and turning of the head to the opposite side. If the stimulation is faint, elevation of the eyelids is the only observable effect. The same authority says that certain clinical facts seem to require the existence of a distinct centre for the levator palpebræ superioris, inasmuch as paralysis may occur limited to this muscle without affecting the other muscles supplied by the third nerve. Ferrier opposes the view of Landouzy that the centre for the levator palpebræ superioris is situated in the angular gyre, holding that, as ptosis does not necessarily accompany lesion of the angular gyre, no causal relationship can be considered as established between the

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<sup>1</sup> Localization of Cerebral Disease, p. 38.

two. He holds that, if the distinct volitional centre for the levator palpebræ superioris is in the human brain, it must be sought for in the region constituted by the bases of the first and second frontal gyres.

"There are, however," he says, "difficulties in the way, for lesions of this region are not uncommon, whereas isolated affection of the levator palpebræ is a comparatively rare occurrence, or at least has not been so obvious as to call for remark. This absence of paresis or paralysis, therefore, requires explanation. It seems to me not improbable that the intimate bilateral association of the oculo-motor nuclei may account for this, and that the escape of the levator palpebræ from paralysis, in cases of unilateral lesion, is simply a higher degree of that which is seen in the orbicularis oculi in facial paralysis of cerebral origin."

It will be noticed in this case that the ptosis was on the same side as the lesion. If not conjugate deviation, there was, at least, some immobility of the eyes. Diplopia also was present for a time. The patient seemed sometimes to have slight right internal squint.

CASE II.—*Tumor of the Optic Thalamus*.—D. M., æt. 19, white, was born in Philadelphia. His father died of bronchitis at the age of 39. He was wild and wayward, not living at home. He was bitten by a horse in the hand during the latter part of June, 1886, and was paralyzed about a week after he was bitten. He was sweeping a store, when he suddenly fell, and on rising, in about five minutes found his right side somewhat paralyzed. He complained of much tenderness over the bowels. He did not suffer with headache before, but his head ached persistently after this time, the pain being most marked over the left eye and round the left temple, though it extended more or less over the whole head.

He had a slight cough, which was worse in the morning, with some muco-purulent expectoration. He had prolonged expiration with crackling râles at the left apex, with a slight impairment of resonance. His right lung was normal. The heart sounds were nearly normal, al-

though the second was somewhat accentuated, and the first slightly muffled. He suffered from anorexia and frequent vomiting.

When first examined, his face was drawn markedly to the left side; he could not wrinkle the right forehead, and there was lagophthalmos of this side. He could not raise his right arm to a level with his shoulder, and was unable to walk well because of paralysis of the right leg. Later, the hemiplegia became profound. He had a blotchy, irregular, erythematous and papular eruption over his chest. His hair had fallen out considerably. The æsthesiometer showed some loss of sensation in the right limbs.

Jan. 18th, 1887.—His pupils were irregular, the left contracted. He was very dull in the mornings and had delusions. His headache was not so severe as before.

19th.—He was considerably brighter. Iodoform ointment was rubbed over the nape of the neck, and three grains given internally. Some improvement was noted in the morning. The external rectus of the right eye was paralyzed. His eyes were bloodshot and his tongue coated. He had no delirium and he took nourishment well.

20th.—His pulse was very rapid, about 160. He complained of much pain in the back of his neck, especially if he moved. He had some difficulty in swallowing. He died on the 21st. His temperature record from January 11th to January 21st most of the time ranged between  $97.5^{\circ}$  and  $100.5^{\circ}$ , oscillating irregularly.

Autopsy (made by Dr. Wm. Osler). His body was that of a poorly nourished lad. There were adhesions of the left pleura. The heart was of average size; on the right side the tricuspid ring was wide and would admit three fingers and a thumb. On the left side the mitral valve was normal, and the aortic was much fenestrated; the fenestrations were large; there were numerous small vegetations. The aorta was smooth. The lungs had a cavity at the left apex. Throughout the upper lobe were numerous caseous masses. In the lower lobe were nu-



merous nodular groups of tubercles. The right lung was crepitant throughout, and presented numerous scattered groups of tubercles. The bronchial glands were slightly enlarged, containing caseous and calcareous masses. The abdominal organs were normal. The dura mater was normal. At the base of the brain the membranes were clear. A little thickening and matting of the membranes were noticeable in the Sylvian fissure, and on full exposure were seen in the pia, covering the convolutions of the island, numerous gray granulations. The ventricles were found much dilated. The amount of fluid was excessive. The septum and fornix were almost diffuent. The posterior cornu was especially dilated. On the left side the thalamus was occupied by a large mass, which projected on the floor of the ventricle, covered by the velum, which was thin and stretched over it. The mass seemed to project from the inner portions of the left thalamus and pushed away the outer part and the caudate nucleus. The mass was grayish-red in color, very regular on the surface, and very firm. It projected forward to within a short distance of the head of the caudate nucleus. A section through the anterior portion of the tumor showed that it had pushed aside the thalamus, and appeared to occupy a portion of its inner half. The gray matter in its vicinity, as well as the internal capsule, was infiltrated and oedematous. On section of the left hemisphere the ganglia seemed normal. On further examination, a few tubercles were found in the anterior perforated space.

One point of interest in connection with this case is the same as one presented by the first case, namely, the existence of complete facial paralysis in association with hemiplegia. The muscles in the distribution of the upper fibres of the facial nerve, namely, the frontalis, orbicularis palpebrarum, etc., were profoundly paralyzed. Commonly, as is well known, in hemiplegia from cerebral lesions—from lesion anywhere above the pons—the upper face escapes, or, if paralyzed at first, promptly regains power. Out of the multitude of hemiplegics, therefore, we have few instances of upper facial paralysis associated

with helplessness of the arm and leg of the same side, although the lower portion of the face is usually affected to a greater or less extent. I have, however, seen several cases in which paralysis of the arm, leg, and the entire face on the same side were present. Some of those cases are on record; a few were collected by Dr. Lloyd and myself, and are to be found in the article on "Tumors on the Brain and its Envelopes," in the "System of Practical Medicine by American Authors," edited by Dr. Wm. Pepper.

The following remarks occur in a paper presented by me to this Society January 23d, 1879 (in this paper was described a case of tumor of the brain situated just in front of the optic chiasm): "A few cases are on record in which the upper fibres of the facial nerve have been paralyzed, as the result of a lesion situated in the brain, above the pons and cerebral peduncles. Chvostek, quoted by Nothnagel (Ziemssen's "Cyclopedia," vol. xii., p. 118), describes a case in which disturbances of speech and a feebleness of the extremities of the left side, present at first, disappeared almost entirely after a few days; on the other hand, the left facial nerve remained almost completely paralyzed (even the branch to the orbicularis palpebrarum), and the autopsy disclosed the presence of a hemorrhagic cyst in the right nucleus lenticularis. The following explanation of the cases of this kind is given by Nothnagel (*op. cit.*, p. 147):

"When the tract of the nerve fibres passing along the base of the nucleus lenticularis, designated as the *ansa peduncularis* (Hirnschenkelschlinge, Gratiolet, Meynert), is involved in the lesion, the character of the symptoms is somewhat different from that described. The most noticeable difference seems to be that in this case the fibres of the facial nerve supplying the frontalis and the orbicularis palpebrarum, which otherwise escape, are paralyzed with the rest, as in the instance reported by Huguenin."

In the present case the upper facial paralysis is probably to be explained, not from the situation of the tumor in the thalamus, but either from the pressure exerted by

it, or the inflammation and œdema adjacent to it, or from both conjointly. In this indirect way both the capsule and lenticular nucleus were involved.

The paralysis of the upper face in the first case must be explained differently. If due to the cortical lesion, the tumor or cyst probably destroyed either the centres for the movements of the eye and forehead, or the white matter constituting the tracts which connect the centres with the parts below. It will be remembered that the lower third of the anterior central or ascending frontal gyre was infringed upon, and that the destruction of white matter extended somewhat beyond the second frontal gyre.

It has occurred to me that the persistence of both the ptosis of one side, and the paralysis in the upper distribution of the facial nerve on the other side, may be explained each from the other. It is supposed that upper facial paralysis and oculo-motor paralysis of cortical origin do not persist, as already stated in quoting Ferrier, because of the intimate bilateral association of the oculo-motor and of the facial nuclei. In this case, supposing the ptosis to have been due to the cortical lesion on the same side, and the facial paralysis of the other side to the same lesion, a curious disassociation was produced, and hence the unilateral symptom in each case persisted.

GLIOMA OF THE RIGHT TEMPORAL LOBE,  
WITH INTERCURRENT HEMORRHAGE. A  
CASE IN WHICH THE QUESTION OF TRE-  
PHINING WAS CONSIDERED AND DECIDED  
AGAINST.<sup>1</sup>

BY CHARLES K. MILLS, M.D., AND GEORGE A. BODAMER, M.D.

THE chief symptoms and conditions in this case were as follows: severe headache, more localized in right temporo-frontal region; pain on localized pressure and percussion; impairment of sight and hearing; choked disk; dilatation of right pupil; three days before death paralysis of left arm and paresis of left leg, paresis of right face, ataxic aphasia.

H. B., æt. 12 years, was admitted to the German Hospital, April 12th, 1887. The father of the patient died suddenly from the effects of a sunstroke; the mother from cholera morbus. The patient was the youngest of six children, and the only survivor, the rest having died in infancy, childhood, or youth, but from what diseases could not be ascertained. The patient was well until she was two years of age, when, according to the statements furnished by a relative, she became wholly paralyzed, and remained so for four weeks, after this slowly ceasing to be helpless, although her gait always continued to be somewhat uncertain and peculiar. She was somewhat sickly for two or three years, when she regained her general health completely. She was sent to school, but was rather dull.

Toward the end of September, 1886, she fell from a

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<sup>1</sup> Read before the Pathological Society of Philadelphia.

front door step, a height of four feet, and was picked up unconscious, in which condition she remained for about ten minutes. In December, 1886, and January, 1887, she suffered from suppurative inflammation of the ear, and, at times, from nose bleeding. After the fall she always complained of headache, which became very intense early in April, 1887, and so continued until the time of her death. When admitted to the hospital, April 12th, her chief complaint was of this headache, which she localized especially over the right temporo-frontal region. Some tenderness to pressure and percussion was present over this same region. Her tongue was thickly coated, but she had no fever or general symptoms. Examination shortly after admission showed both pupils somewhat dilated, the right, however, undoubtedly larger than the left. She had some difficulty in seeing, and her hearing was somewhat defective, or, at least, it was difficult, at times, to fix her attention, or get responses without loud questioning. Unfortunately, no detailed study of sight, hearing, or the other senses, was made. At the time of admission to the hospital she gave no evidences of paralysis, or of any other symptoms than those above detailed.

Until the evening of Friday, April 29th, her condition continued as above described. At this time, she suddenly became paralyzed in the left arm, and paretic in the left leg; her speech was also affected, articulation becoming for a time so indistinct and imperfect that nothing she attempted to say could be understood. She did not become completely unconscious at the time of the paralytic attack; she did not have either Cheyne-Stokes or stertorous breathing. Her mouth was slightly drawn to the left, her pupils remaining dilated, the right continuing larger than the left. So far as could be determined, sensation was not affected on either side. Ophthalmoscopic examination showed choked disk in right eye. The condition in the left eye was not positively determined, although the vessels were engorged.

On May 1st a consultation was held to consider the

question of trephining for the removal of the supposed growth. At the consultation were present Drs. Adam Trau, D. Hayes Agnew, F. H. Gross, J. B. Deaver, and Charles K. Mills, with the resident physician of the hospital, Dr. George A. Bodamer. After some discussion, it was concluded best not to trephine. This conclusion was reached because of the presence of certain symptoms and the absence of others, which threw doubt upon the exact location of the growth. Apparently, at first sight, the case was one of brachial or brachio-crural monoplegia, from lesion of the arm centres of the cortex and adjacent areas. The paralysis of the arm and paresis of the leg were the most marked conditions. The patient, however, had also dilatation of the right pupil, and either paresis of the lower right face, or slight spasm of the lower left face. It was really somewhat difficult to determine whether the condition was one of paresis on one side, or very slight spasm on the other. The left corner of the mouth was slightly drawn up, the right apparently drooping somewhat. It was argued that if the condition was one of right-sided facial paresis, the paralysis of the limbs associated with this condition could not be explained by a single growth of the cortical centres of the right side; neither was the dilatation of the right pupil easily explicable from the standpoint of a lesion of the arm and leg centres of the cortex. The entire absence of spasm, either local or general, was also somewhat against the probability of the tumor being one so superficially localized in the motor zone, and connected with the membranes as to be readily reached by operation. Finally, the sudden occurrence of marked paralysis, after the long continuance of other symptoms of brain tumor or abscess, showed that either a hemorrhage had occurred, as was suggested by Dr. Trau, or that a sudden and somewhat extensive break-down of tissues in the vicinity of the old lesion had taken place. The suddenness of the paralysis seemed to negative the view that it was the result of the extension of the growth by slow development from a latent to the motor region, although this idea was con-

sidered, on the whole, the conditions seemed to point to a growth either at the base on the right side, or so low down in the cerebrum as to exert pressure upon the descending motor tracts and, possibly, also upon some fibres of the facial and oculo-motor nerves of the same side. The conditions as to consciousness and speech were peculiar. The patient certainly understood much, if not all, that was said to her on May 1st, although the voice had to be somewhat loud and the manner earnest in order to get response. Her speech was thick and indistinct; the aphasia was ataxic rather than amnesic. On asking her what her name was, she answered "Hattie," so that she could be understood, although the articulation of the word was thick and imperfect. The patient died the morning of May 2d.

*Autopsy.*—At the autopsy, which was held thirty-six hours after death, were present Drs. Adam Trau, F. H. Gross, Charles K. Mills, and George A. Bodamer. The skull was found to be unusually thin, and roughened in a peculiar way on its inner surface; and here and there were jagged processes, especially in the occipital region. A considerable area of the calvarium was injected; and the outer surface of the dura mater showed corresponding streaks and patches of a bright red color, markedly in the postero-frontal, parietal, and occipital regions. The appearances were present on both sides, but were much more decided on the right. When the dura mater was removed, the exposed pia mater and surface of the brain at first showed nothing abnormal, except here and there a few spots of unusual coloration. Evidently no growth connected with the membranes was present on either the median or lateral aspects of the brain.

The removal of the brain from the skull was conducted with great care, but during the process a slight break through the surface of the brain occurred at a point about the middle of the right second temporal gyre, and through this poured out a mahogany-colored fluid. The tear enlarging, a cavity and growth were revealed. The neoplasm was a frangible, semi-solid, light purplish mass, hav-

ing beneath and partly around it a cavity containing detritus, and a large unorganized, and evidently recent, clot. The space occupied by the neoplasm, cavity, and clot was irregular in shape, its greatest length being about three inches, and its height and width probably about one-half its length. It occupied a large portion of the interior of the right temporal lobe, but was strictly limited to it. Careful sections showed that the anterior one and one-half inch of the lobe was not involved, and the tissue destroyed did not extend quite to the temporo-occipital junction.

A microscopical examination was made by Dr. Bodamer. This revealed a hemorrhagic or rather a very vascular glioma.



## A SECOND CLINICAL STUDY OF HEMIANOPSIA. CASES OF CHIASM-LESION. DEMONSTRATION OF HEMIOPIC PUPILLARY INACTION.<sup>1</sup>

BY E. C. SEGUIN, M.D.

I BEG leave to present to the Society notes of three cases of hemianopsia of the peripheral or neural form, probably caused by a lesion of the optic chiasm. None of these cases are completed by autopsy, yet I think that the diagnosis is clear enough to render them worthy of record, and to make them the subject of a few clinical and diagnostic remarks.

All of these cases present the remarkable pupillary light reaction, first indicated by Von Graefe, and designated by Wernicke as hemiopic pupillary reaction. One of the patients is present this evening, and upon him I may be able to demonstrate this rare and valuable symptom, which, to the best of my knowledge, has not yet been observed (or at least recorded) in this country.

I shall append a series of diagnostic propositions bearing upon all varieties of hemianopsia, which, though far from perfect, may perhaps prove useful for our further study of this symptom.

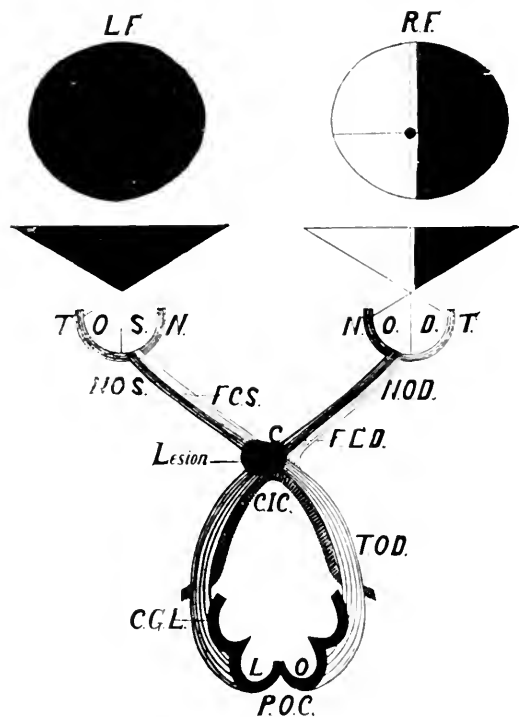
*CASE I.—Loss of vision in left eye ; temporal hemianopsia with right eye. Complete atrophy of left optic nerve ; partial of the right. No other symptoms of cerebral lesion. Hemiopic pupillary inaction.*

H. S., a grocer's clerk, a German, 20 years of age, presented himself at the Manhattan Eye and Ear Hospital, service of Dr. David Webster, on October 26th, 1887. Dr. Webster kindly referred him to my department for observation and treatment.

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<sup>1</sup> Read before the New York Neurological Society at the stated meeting held Dec. 6th, 1887.

Health has been fairly good, except that for six years he has had occasional attacks of bilateral headache, mostly frontal, often accompanied by nausea and vomiting. This was apparently migraine. Headache not in family. In the last two years more constant frontal pain. No injury or syphilis. Two years ago, the patient accidentally discovered that his left eye was partly blind; he could see only large objects. This has since progressed to total loss of vision. In the last few months less headache, without vomit-



Case 1 H.S.  
1887

Chiasm Hemianopsia,

LF, left visual field; RF, right visual field; OS, left eye; OD, right eye; N nasal; T, temporal halves of eyes; NOS, left optic nerve; NOD, right optic nerve; FCS, left fasciculus cruciatus; NLD, right fasciculus cruciatus; C, chiasm; TOD, right optic tract; CIC, commissura inferior cerebri (non-optic fibres of chiasm); CGL, corpus geniculatum laterale; LO, lobus opticus, which together make up POC, the primary optic centres. For central parts of optic apparatus cf. fig. on p. 35 of this JOURNAL for Dec., 1886.

ing. No other symptoms, sensory or motor. States that he smells and tastes well. Dr. Webster found L. V = 0; R. V = 2%. The right visual field showed temporal hemianopsia, as represented in above diagram.

The left optic nerve is fully atrophied, the right partly. Examination shows that neither pupil responds when light is thrown into the (blind) left eye. When the light is thrown into the right eye, contraction occurs in both pupils; showing that centripetal conduction is preserved in the right optic nerve to the lobus opticus, and that centrifugal conduction is perfect through both motor oculi nerves to irides.

The right pupil apparently reacts well to light. As usual, the vertical division line between the light and dark half-fields of that eye falls outside of the point of fixation. No paralysis of ocular muscles or of any part of the body. In short, there are no other symptoms of cerebral or spinal disease. General health fairly good.

Dr. Webster and I considered the case as one of chiasm-lesion, involving three-fourths of the decussation, *i. e.*, destroying both fasciculi of the left optic nerve and the fasciculus cruciatus of the right eye. That the lesion is in the peripheral or basal part of the optic apparatus is indicated by the loss of pupillary reflex in the blind left eye, and by the extreme degree of atrophy present. By the help of the above diagram of the decussation the probable seat of the lesion is made clear.

That the lesion does not involve one optic tract caudad of the chiasm is certain, because in that case there would be lateral or homonymous hemianopsia in both eyes.

In this case, a search was made for hemiopic pupillary inaction in the right eye, with the following results, which I demonstrated to Dr. Webster and a number of the staff of the hospital, and some other physicians. When the light from an ophthalmoscopic mirror was thrown directly into the right pupil, a good reaction was at once obtained in the two irides.

When the beam of light was made to strike the cornea from the nasal side (the side with vision) at any angle, a reaction was also obtained all around the pupil in both eyes.

When, however, the beam of light was made to enter the right eye from the temporal or dark side at an angle of  $60^{\circ}$ , or rather less, striking chiefly the nasal or anæsthetic half of the retina, no reaction, or almost none, occurred in the left pupil (and in the right). Therefore, we had before us a striking example of hemiopic pupillary reaction, or *inaction*, as I would prefer to call it. An interesting fact is that the entire iris responds to the stimulus applied to the temporal half of the retina and only transmitted centripetally by the fasciculus lateralis of the right eye.

That the whole muscular apparatus of the iris (sphincter or constrictor pupillæ) should contract from a reflex action originating in one half of the retina is easily understood if we remember that the ciliary nerves go to form a plexus containing ganglion cells in the iris. With such a nervo-muscular apparatus, the motor impulse must of necessity be diffused throughout the entire iris, and not restricted to one of its halves, or a smaller part.

November 21st.—Re-examined for hemiopic pupillary inaction. Same result obtained, viz., when the beam of light is thrown into the right pupil from any angle on the nasal side, or from  $90^\circ$  directly into the centre of the retina, or to the temporal (blind) side as far as  $70^\circ$  or  $60^\circ$  on an equatorial arc, a good pupillary reaction is obtained. When, however, the ray of light is made to enter more obliquely from the temporal side, from  $60^\circ$  to  $40^\circ$  on the equatorial arc, no reaction was obtained in the eye examined or in the left blind eye.

Both pupils contract under accommodative effort.

L. V = 0; R. V =  $\frac{6}{200}$ . With the right eye can just make out No. XIV. Jaeger at 8 inches.

CASE II.—*Partial blindness, with marked atrophy of both optic nerves. Bi-temporal hemianopsia, plus obscuration of upper nasal quadrant of left field. No other symptoms of cerebral disease. Hemiopic pupillary inaction in right eye.*

J. H., a male, aged 41 years, was referred from the ophthalmic department (service of Dr. D. Webster) to the nervous department of the Manhattan E. and E. Hospital as an interesting case of atrophy of optic nerves probably due to intra-cranial disease.

The patient stated that since the spring of 1886 there has been steadily increasing failure of vision in the left eye, to almost complete blindness. Occasional frontal headaches, never very severe. Has had diplopia at times, which he describes quite intelligently, in a rather peculiar way, stating that where there were two objects he would see three, where there were four he would see five. For several years occasional slight attacks of vertigo. No other symptoms indicating cerebral or spinal disease.

Denies syphilis and injury to head.

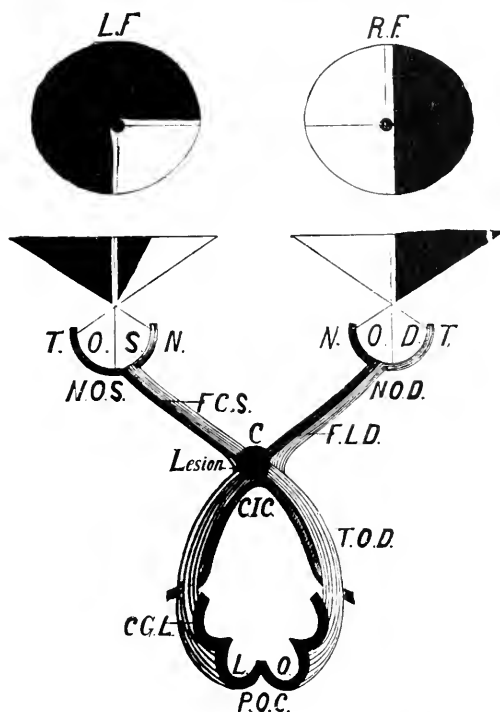
Examination shows: Ocular muscles normal. Pupils moderately dilated and responsive to light. Both optic nerves appear considerably atrophied. Visual fields, as per subjoined diagram (blackboard test).

This peculiar form of hemianopsia (nearly typically bi-temporal) would indicate the existence of a lesion so placed as to destroy both fasciculi cruciati, and the basal or inferior half of the fasciculus lateralis of the left eye.

Nov. 11th. Re-examined. Fields (tested on blackboard) are as before, except that in the field of the right eye the vertical line of separation now passes through the point of fixation. Both pupils contract with accommodative effort. Both pupils independently show reaction to light; barely perceptible in left eye, marked in right eye; slight but distinct transferred or consensual light reaction from one eye to the other. Right eye exhibits hemiopic pupillary inaction. When light is thrown into the pupil from the temporal side up to  $60^\circ$  no reaction whatever takes place; but when the light pencil is thrown in from the nasal side at any angle and past the optical axis ( $90^\circ$ ) to about  $60^\circ$  on the temporal side, distinct iridic contraction occurs. The reaction is obtained

equally well in these meridians from any degree above or below the equatorial line.

Left pupil reacts slightly to central illumination, but tests on either side yield no trustworthy or definite reactions.<sup>1</sup>



*Casell. S.H.*  
1087

*Chiasm Hemianopsia.*

L.F., left visual field; R.F., right visual field; OS, left eye; OD, right eye; N and T, nasal and temporal halves of each retina; NOS, left optic nerve; NOD, right optic nerve; FCS, left fasciculus cruciatus; FLD, right fasciculus lateralis; C, chiasm; CIC, commissura inferior cerebri (non-optic fibres of chiasm); TOD, right tractus opticus; CGL, corpus geniculatum laterale; LO, lobus opticus or anterior group of corpora quadrigemina, which together make up POC, the primary optic centres.

<sup>1</sup> Re-examined Dec. 3d. Condition as before except that the optic nerve atrophy has progressed, and is more evident in right nerve. The right visual field is exactly hemianopic; the left field shows a further geometric loss; one-third (the inferior) of the lower nasal quadrant now being dark. The pupils are larger and nearly inactive. A faint but distinct hemiopic pupillary inaction is obtained when the right eye is tested as above described. V. R. =  $\frac{2}{80}$ , V. L. =  $\frac{2}{80}$ .

This result is interesting as showing that the geometric amount of field-reduction is not the only factor in loss of vision.

CASE III.—*Almost total loss of vision in the right eye. Temporal hemianopsia with left eye. Partial atrophy of both optic nerves. Distinct hemiopic pupillary inaction. No symptoms of cerebral disease.*

A. C., an Italian barber, twenty-five years old, was referred to Drs. Agnew and Webster and to me by his physician, Dr. Rufus Baker, of Middletown, Conn. I saw him October 10th, 1887.

Some two years ago vision gradually became impaired. The left eye improved (?), but the right became progressively worse to complete blindness. No diplopia. In the last two months the patient has become aware of hemianopsia. No other symptoms, except some bilateral temporo-frontal headache in last two weeks only. General loss of strength. Smell and taste preserved; no hallucinations. No epileptiform or syncopal attacks.

No etiology can be made out; no injury to head or syphilitic infection.

Examination. No paralytic symptoms or anæsthesia. Equilibrium good; grasp: right hand  $23^{\circ}$ , left hand  $20^{\circ}$ . No ataxia. Patellar reflex normal.

Ocular apparatus. Muscles act well. Pupils are equal and active, directly and in association. Vision is quite lost in right eye. Left eye shows regular temporal hemianopsia, the vertical line passing distinctly outside of point of fixation.

The ophthalmoscope shows moderate atrophy of the optic nerves, much more marked in the right eye. Vessels are, however, full in both eyes. No retinal changes.

Re-examined November 20th, 1887, in presence of Drs. Baker and Hallock, at Middletown. The patient has taken iodide of potassium up to 75 grains three times a day. About two weeks ago found that he had some sight with the right eye upward. Left eye unchanged. Present condition: Pupils normal, medium and mobile. They contract independently by central illumination, and also in association. No distinct hemiopic inaction can be obtained in the right eye, though pupillary action seems quicker and fuller when the ray of light concentrated by a concave mirror is thrown into the pupil from above on the right or left of vertical meridian. In the left eye, typical hemiopic pupillary inaction is obtained: *i. e.*, when light is thrown obliquely into pupil from the temporal or dark half-field no reaction occurs. When the ray of light is brought nearly (at  $60^{\circ}$  or  $70^{\circ}$ ) in the pupillary axis, in it, or carried beyond into the nasal half-field, a full and quick pupillary reaction occurs.

Vision. With the right eye a large white object, such as one's hand, or a piece of paper, is perceived in a part of the two upper quadrants of the field. The two quadrants are separated by a dark strip vertically placed in the field, as shown in annexed diagram. In the left eye there is temporal hemianopsia as before, with vertical line (not quite straight this time) passing outside of



in which the hemianopsia is produced by a central or cerebral lesion, situated anywhere in the optic apparatus caudad of the primary optic centres.

That the peripheral lesion in the cases here related is not one affecting one optic tract is shown by the form of H., *i. e.*, in its not being of the lateral or homonymous form. The lesion is quite surely at the chiasm itself, situated as approximately represented in the diagrams which accompany each case.

There are several points of interest in the semeiology of these cases, but I shall occupy the time of the Society only by the consideration of one symptom presented by all three patients—a relatively new symptom, and one which I believe has great diagnostic value, enabling us to decide with nearly positive exactness whether a hemianopsia is due to a central or to a peripheral lesion. I refer to the symptom called by Wernicke<sup>1</sup> hemiopic pupillary reaction, and which I now propose to designate, more accurately perhaps, *hemiopic pupillary inaction*.

Allow me to clearly state the nature of the symptom and the conditions of its demonstration.

The normal optic apparatus contains a reflex arc that automatically regulates the size of the pupils under different degrees of illumination. The component parts of this arc are: the whole retinal expansion (with special sensitiveness of the macula) as a receptive organ for light, the optic nerves and tracts as centripetal channels of transportation of the impulse produced by light to the anterior group of the corpora quadrigemina (the lobi optici) where a reflection takes place to the nuclei of the motor oculi nerves (to both nerves at once). The mechanism and the physiology of this part of the action is unknown to us. From the nuclei of the motor oculi a centrifugal or motor

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<sup>1</sup> C. Wernicke, "Ueber hemiopische Pupillenreaction." *Fortschritte der Medicin*, I. Heft 2, 1883.

I owe an apology to Professor Wernicke for having misquoted and perverted the true sense of his article in a brief reference which I made to it in my paper on the Pathology of Hemianopsia of Central Origin. It is incomprehensible to me now how I could have failed to comprehend Wernicke's paper on first reading it.



impulse goes out to the ciliary nerves and to the iris muscles, producing contraction. A luminous impulse from one retinal expansion produces the reflex motor act in both irides (associated or consensual pupillary reflex), and the entire iris contracts in all instances because the termination of the ciliary nerves in the irides is plexiform, with intercalated ganglion cells in the muscle.

If a ray of light be made to enter the eye directly in its optical centre, striking the macula lutea, the pupillary reaction is quickest and most complete. If now the light be so moved as to cause the ray to impinge from various angles above or below, nasalward or temporalward from the optical axis, pupillary reaction still occurs.

In pathological cases, the reflex arc may be broken at various points. (a) The entire retina or one of its halves, or a more limited part of it, may be insensitive to light through local disease or neural degeneration. (b) The conducting centripetal paths (optic nerves), may be interrupted by a lesion; in these categories belong our cases of hemianopsia with defective pupillary reaction. (c) The centre for the reflex act, the gray matter in which the mysterious transformation of a sensory into a motor impulse takes place, which, according to most recent researches (von Gudden and others) is in the *lobi optici*, may itself be diseased. (d) The conducting centrifugal paths (motor oculi and ciliary nerves) may be interrupted by a lesion, or the nuclei of the motor oculi may be diseased. (e) The terminal motor organ, the iris, may be rendered rigid or inactive by local disease (iritis). This general statement will serve to explain all varieties (pathologically speaking) of diminution or loss of pupillary reflex.

In the variety to which I desire to ask your attention this evening, viz., *hemiopic pupillary inaction*, one-half of each retina being physiologically inert, or anæsthetic, fails to receive any impulse from the light which is thrown on it. All three cases related, including the patient upon whom I shall attempt a demonstration, present this symptom.

But certain restrictions must be placed upon the meaning of the word hemiopic as applied to the results of testing, for we find the facts as follows in these cases:

If, in the hemiopic eye, we throw the pencil of light (concentrated from a lamp by an ophthalmoscopic mirror) directly into the eye in its optical axis, we obtain a full and quick pupillary reaction. If, now, we move the mirror more and more nasalward from the optical axis, and throw the beam of light through the pupil upon the nor-

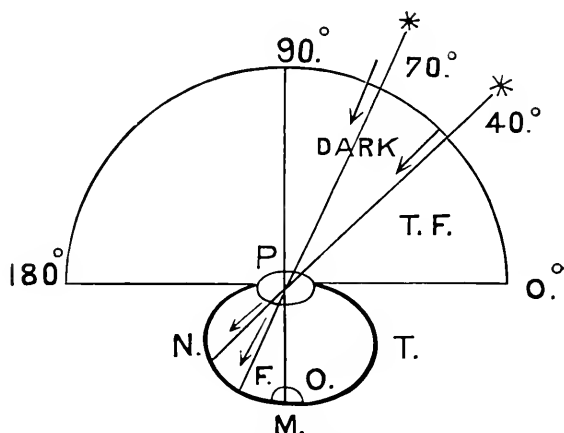


FIG. 4.—Diagram illustrative of test for *hemiopic pupillary inaction*. The lines represent a horizontal plane through the left eye and its visual field. FO, fundus oculi; M, macula lutea; N, nasal half of field which is anæsthetic in temporal hemianopsia; T, temporal half of retina; P, pupillary aperture. 180° to 0°, the equatorial arc or semicircle. 90°, vertical point and line passing through centre of eye to M. 70° and 40°, rays of light striking the insensitve nasal half of retina, producing no pupillary reflex. About 6° is a fair average for the angle obtained in the various tests of the three cases.

mal (temporal) half of the retina, a good reaction occurs until the light ceases to enter the pupil. If we next move the mirror outward or temporalward from the optical axis of the eye, we obtain pupillary reaction over quite an arc of the circle; but after passing a point between 60 or 40 degrees from the horizontal pupil line (see Fig. 4), pupillary reaction no longer takes place, and we have a demonstration of hemiopic pupillary inaction. On first thought, it would seem fair to conclude from this experiment that only the peripheral 40 or 60 per cent of the

nasal half of the retina is insensitive or anæsthetic, but this is not so. The reason why the light experiment is not as geometrically exact as the perimetric test is, that it is mechanically impossible to throw a beam of light so that it shall be focussed only on very limited portions of the retina. Even with a small pencil of light there is more or less illumination of the entire pupillary aperture and diffusion of light within the eye. If we could exactly restrict the action of the ray of light, the pupillary immobility would be evident the moment the mirror was so displaced temporalward (in temporal hemianopsia) as to strike the retina a little to the nasal side of the macula.

It follows that in testing for hemiopic pupillary inaction we must observe a number of precautions. My own plan of procedure is as follows: The patient being in a dark or nearly dark room, with the lamp or gas light behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus, upon the eye and note the size of the pupil. With my other hand I now throw a beam of light, focussed from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all the angles of incidence of the ray of light. With a little practice this can be thoroughly done in a few moments. In testing the pupils for other purposes (in cases of tabes, dementia paralytica, etc.) by ordinary daylight or lamplight, it is also important to make the patient look at a distant object in order to relax accommodation, and thus exclude accommodative iris movements.

Accommodative pupillary movements were preserved in all three cases.

The reasons why I have treated at such length of the symptom hemiopic pupillary inaction are: its intrinsic value for diagnosis, as will be shown farther on; and the

fact that, to the best of my knowledge, it has not been observed, or, at least, publicly referred to in this country.

In conclusion, I wish to append, as a sort of conclusion to my three papers on hemianopsia, a series of diagnostic propositions or laws, applicable to all cases presenting this symptom. It is far from my thoughts to claim that these propositions are final or absolutely exact in all particulars. They will doubtless be amended, but perhaps in their present shape they may temporarily prove useful to the practical physician.

#### FIRST CATEGORY OF CASES.

Vertical or quadrant hemianopsia alone; or, at least, without other special symptom of organic intra-cranial lesion.

1. Homonymous or lateral H. The lesion is either in the cuneus of the hemisphere opposite the dark half-fields, or it involves the tractus opticus opposite to the dark half-fields.

*a.* When the lesion is central, *i. e.*, involving the cuneus and adjacent basal gyrus, the optic nerves appear normal to the ophthalmoscope, central vision is good and the pupillary reflex is fully preserved. Except: When the lesion is a very large tumor, there may be neuro-retinitis, and, after a long time, atrophy of both optic nerves, with blindness and total loss of pupillary reflex. At no stage of the disease is hemiopic pupillary inaction observed.

*b.* When the lesion involves the tractus opticus, there is, from an early period, hemiopic pupillary inaction and partial optic nerve atrophy. Later in the course of the disease, neuro-retinitis of both eyes may occur, and be followed by atrophy, total blindness, and by pupillary immobility.

*c.* Homonymous quadrant obscuration. If, for example, the lower lateral quadrant of each visual field be obscured, the lesion involves only the basal half of the cuneus of the opposite side. (This proposition is justified by Hun's case, vide *The American Journal of the Medical*

*Sciences*, Jan., 1887, pp. 141-144). In such a case there would be full pupillary reaction.

II. Heteronymous H. is present, usually, with hemiopic pupillary inaction, and atrophy of the optic nerves.

*a.* Bi-temporal H. in both eyes; *i. e.*, the temporal or external half of the visual field of each eye is obscured: optic nerve atrophy and hemiopic pupillary inaction are present. The lesion is at the chiasm, in its median part, so placed as to destroy both fasciculi cruciati.

$\alpha$ —There may be temporal H. in one eye, and complete loss of vision with atrophy of the optic nerve and pupillary immobility in the other eye. The lesion is at the chiasm, so placed as to destroy both fasciculi cruciati and one fasciculus lateralis (Case I.).

$\beta$ —There may be lateral H. of one eye and obscuration of three quadrants of the field of the other eye. The lesion is then at the chiasm, so situated as to destroy both fasciculi cruciati, and the ventral or dorsal half of one fasciculus lateralis (Case II.).

$\gamma$ —With lateral H. of one eye, there may be a superior, partial, or quadrant obscuration of the other eye, as in Case III. at the last examination. In such a case, or in a similar one of irregular quadrant obscuration (not strictly lateral or homonymous), the lesion is an infiltrating one, affecting various bundles of the chiasm and adjacent optic nerves in an irregular way.

*b.* Bi-nasal H. is exceedingly rare. To my knowledge there is only one case on record with a post-mortem examination. There should also be in this form hemiopic pupillary inaction, and atrophy of the optic nerves. The lesion is at the chiasm, so placed as to injure both fasciculi laterales. In Knapp's case,<sup>1</sup> calcified and enlarged carotid arteries compressed the sides of the chiasm. A tumor might possibly so grow as to act in the same manner.

*c.* Superior or inferior H. This form is rarely due to a neural lesion. When present with a somewhat irreg-

<sup>1</sup> "Hemiopic and sector-like defects in the field of vision." Brown-Séquard and Seguin's *Archives of Scientific and Practical Medicine*, 1873, No. 4, page 308.

ular demarcation line, it is due to embolism of one of the main branches of the central artery of the retina. A lesion might theoretically be so placed at the chiasm as to injure its ventral or dorsal aspect only, thus causing the horizontal H., but we have no positive knowledge of such a condition.

*d.* Monocular H. of any form is due to a lesion of the optic nerve frontad of the chiasm, or to a lesion on one side of the chiasm. If not exactly geometric and without atrophy of the optic nerve, its cause is probably within the eye.

*e.* A few cases of rapidly varying forms of H. in the same subject have been described. A remarkable one has been recorded by H. D. Noyes and T. A. McBride.<sup>1</sup> The pathology of such cases is unknown; possibly, changing states of circulation and nutrition in the cuneus would explain the symptoms.

#### SECOND CATEGORY OF CASES.

Vertical or quadrant hemianopsia coinciding with other symptoms indicating definite cerebral disease.

I.—Lateral or homonymous H. in such combination.

*a.* Co-existing with hemianæsthesia and choreiform or ataxic movements of one-half of the body (of the limbs on the same side of the body as the dark half-fields), without marked hemiplegia, it is probably due to a lesion of the caudo-lateral part of the thalamus, or of the caudal division of the internal capsule on the side opposite to the dark half-fields.

*b.* Lateral H. with complete hemiplegia (spastic after a few weeks) and hemianæsthesia, is probably caused by an extensive lesion of the internal capsule in its knee and caudal part.

*c.* Lateral H. with common or typical hemiplegia (spastic after a few weeks), aphasia, if the right side be paralyzed (right lateral H.), and with little or no anæsthesia,

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<sup>1</sup> Henry D. Noyes, "Acute Myelitis with Double Optic Neuritis," Knapp's Archives of Ophthalmology, IX., p. 199, 1880.

is quite certainly due to an extensive superficial lesion of the area supplied by the middle cerebral artery. We would expect to find, as in Westphal's case,<sup>1</sup> softening of the motor zone and of the gyri lying at the extremity of the fissure of Sylvius, viz., the inferior parietal lobule, the supra-marginal gyrus, the gyrus angularis, and the subjacent white substance. Embolism or thrombosis of the Sylvian artery would be the most likely pathological cause of the softening.

*d.* Lateral H. with moderate loss of power in one-half of the body, especially if associated with impairment or loss of muscular sense, would probably be due to a lesion of the inferior parietal lobule and the gyrus angularis with their subjacent white substance, penetrating deeply enough to sever or compress the optic fasciculus on its way caudad from the primary optic centre to the visual half-centre (the cuneus opposite to the dark half-field).

In all the above symptom-groups of the second category there would be full pupillary reaction and no atrophy of the optic nerves, unless choked disk has occurred and obscured the phenomena. Even with complicating choked disk, there should not be hemiopic pupillary inaction previous to the setting in of atrophy.

*e.* Lateral H. with other symptoms indicating lesion of the primary optic centres on one side (viz., the corpus geniculatum laterale and one lobus opticus) should be here considered, but our present knowledge is not such as to enable us to recognize disease thus situated. There would certainly be, besides lateral H., hemiopic pupillary inaction, and early atrophy of the optic nerves, as the primary optic centres contain a centre for reflex movements and a trophic centre for the optic nerves. We might theoretically suggest that there would be disorders of sensibility, and perhaps choreic-ataxic movements of the limbs on the same side as the dark half-fields.

*f.* Lateral H. with hemiopic pupillary inaction and with

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<sup>1</sup> C. Westphal: "Zur Localisation der Hemianopsie und des Muskelgefühls beim Menschen." *Charité-Annalen*, viii., p. 466, 1882.

paralytic symptoms belonging to the class known as crossed paralysis would indicate a lesion at the base of the brain, upon the crus cerebri, injuring the tractus opticus opposite to the dark half-fields, and adjacent structures. The most probable combination would be paralysis of the third, fourth, and sixth cranial nerves on the side of the normal half-fields (one or all of these nerves); with partial hemiplegia, without anæsthesia, on the same side as the dark half-fields, caused by injury to the crus cerebri. An extensive lesion thus placed might also affect the trigeminus. In a fully developed case of this group, we would have as direct symptoms paralysis of the ocular nerves, and perhaps of the trigeminus; as crossed symptoms, lateral hemianopsia, with hemiopic pupillary inaction and early atrophy of the optic nerves; also partial paralysis without anæsthesia on the same side of the body as the dark half-fields. In some cases, choked disks would appear early without interfering with the hemiopic symptoms; at least not until general atrophy of both optic nerves and complete blindness set in.

11.—Heteronymous H. with other symptoms of disease at the base of the brain.

*a.* Bi-nasal H. with paralysis of all, or nearly all, of the motor and sensory nerves of both eyes, would indicate a lesion inclosing the chiasm, spread out laterally from it so as to affect the orbital nerves, leaving the central (decussating) fibres of the chiasm intact.

*b.* Bi-temporal H. might be associated with unilateral or bilateral anosmia, thus indicating disease in the medio-frontal edge of the chiasm, extending frontad upon the orbital roof.

*c.* Bi-temporal H. in one eye, with complete blindness of the other, and paralysis of some or of all the nerves which enter the orbit of the blind eye would indicate a lesion laterad of the chiasm involving the orbital nerves, the optic nerve, and three-quarters of the chiasm (as represented in the diagram of Case I.).

In symptom-groups *a* and *b* there would be hemiopic pupillary inaction, with partial atrophy of both optic



nerves; the associated or consensual pupillary reaction, as well as accommodative pupillary contraction, might be preserved.

In symptom-group *c* there would be hemiopic pupillary inaction in one eye, and total loss of direct pupillary reflex in the other (blind eye). Consensual pupillary contraction would not take place when the light was thrown into the blind eye, but the reverse would be true provided that the motor oculi nerve of the blind eye were not paralyzed.

All these symptom-groups, again, may be complicated with choked disk, though this rarely occurs.

Lastly, more irregular grouping of symptoms of this category, not explicable by any of the above diagnostic propositions, would quite certainly be due to the presence of multiple lesions (in syphilitic subjects especially).

## Clinical Cases.

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### REPORTS OF CASES OF INSANITY FROM THE INSANE DEPARTMENT OF THE PHILADEL- PHIA HOSPITAL.

PREPARED UNDER THE DIRECTION OF CHARLES K. MILLS, M.D., VISITING  
PHYSICIAN.

#### *Cases of Insanity Occurring at Puberty and Adolescence, with Remarks.<sup>1</sup>*

That certain physiological periods—eras of development, of climax, or of decline—predispose to insanity of some form is almost universally admitted by those who have practical experience with the insane. Some of the classifications of insanity, those of Spitzka and Clouston, for instance, have types or classes of mental disease based upon this view. Spitzka, among his pure insanities, has two forms which he regards as attacking the individual in essential connection with the developmental or involutonal periods, namely, insanity of pubescence or hebephrenia, and senile dementia. Clouston discusses an insanity, or rather, the insanities of puberty and adolescence, a climacteric insanity, and a senile insanity. Many difficulties beset the attempt to erect a typical form of insanity on such bases. One of these springs from the fact which is constantly impressing itself upon every working neurologist and alienist, that any and every form of mental disease may occur in each of these periods. This fact has led some to doubt and discredit such classifications. In old age, mania, melancholia, paranoia, epileptic insanity, parietic dementia, and other types of insanity are sometimes developed. At the climacteric period in women, so diverse are the forms of insanity which present themselves for investigation or treatment, that it is scarcely worth while to discuss the point. Considerable experience has, nevertheless, inclined me, not without hesitation, to accept the view that a special type of senile dementia exists which can be differentiated from other senile insanities; and I incline, but also

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<sup>1</sup> These cases were shown and the remarks were made to the students of the Fourth-Year Course of the Medical Department of University of Pennsylvania.

with some hesitation, to the view of Clouston, that the climacteric period stamps certain peculiarities upon the mental disorder which develops in some cases, so as to give a recognizable type—a sub-acute psychosis with special features.

The hebephrenia of Hecker is certainly hard to differentiate from some mental disorders occurring at the period of puberty and adolescence, and equally easy to separate from other insanities occurring at the same period. Of the forms difficult to differentiate, katatonia and the masturbational insanity of Spitzka might be cited; of those comparatively easy to separate, delusional melancholia and common acute mania might be mentioned. I have seen not a few cases of profound melancholia, delusional and semistuporous, occurring between puberty and adolescence. Only a short time since, for example, I had in charge a young girl with typical melancholic depression, and delusions of pregnancy and of religious depression, who was also markedly suicidal, but who soon recovered on a tonic treatment with full feeding. A few years ago, a beautiful and intelligent girl of seventeen was sent to me from a distance. I feared that the case was one of ordinary hebephrenia, feared this because then the outlook would not have been good, many of these cases passing into a condition of dementia. This patient was excited, exhilarated, full of action, with, in short, the ordinary evidences of mania, combined with a tendency to a sort of romanticism or sentimentalism. In reading a novel she would transfer the characters in the book to persons in real life with whom she was acquainted. She was wanting, however, in other special features of typical insanity of pubescence. She recovered in a few months under treatment, conium having been the chief medicinal agent employed.

Before presenting a few illustrations of insanity occurring at the period of puberty and adolescence, it might be well to say a few words in a general way about this period. Can we distinguish, so far as special mental affections are concerned, between cases occurring at puberty and at adolescence? This, I think, is doubtful. Spitzka, in defining hebephrenia, speaks of it as "coinciding with or following the period of puberty," that is, practically as occurring at puberty or in adolescence. Puberty is the time when the precreative and reproductive powers are initiated; adolescence is the growing period immediately following puberty. The French law makes the period of puberty fourteen years in the male, and twelve years in the female. Adolescence, as commonly recognized, extends from fourteen years in the male, and from twelve in the female, up to twenty-five in the male, and anywhere from twenty-one to twenty-five in the female. The French speak of pubescence, adolescence, and nubility. Nubility is the completion of adolescence; it means marriageability in the physiological sense; it is the culmination of adolescence. Hebephrenia is then practically the insanity either of puberty or adolescence. We do not see many cases of insanity at the age of twelve to fifteen years,

but we do see many between the ages of fourteen and twenty-five years, both in the male and female. If adolescence is divided into two stages, as it may properly be, more cases will probably be found to occur in the latter half.

Some of the cases now to be reported will be found to measure pretty well up to the description of hebephrenia which, according to Spitzka, is "characterized by mental enfeeblement, marked by a silly disposition, following a preliminary period of depression, which has the same tinge as, without the depth of, that characterizing melancholia." Others do not as well correspond to this description, and yet cannot well be otherwise classed.

#### CASE IX.—*Hebephrenia in the Female.*

Reported by Dr. Harriet Brooke, Assistant Physician.

J. H., aged 17 years, born in Ireland, a Roman Catholic, domestic, was admitted to the Hospital August 21st, 1886. She is of medium height, with dark brown hair and eyes. Her expression and manner are intelligent, and her head normal in conformation. She appears well nourished and is of sanguine temperament, very quick to resent anything that does not meet her approval; she can read and write. Her memory is good.

Her father is dead, and it is said that there was no insanity in his family. Her mother is living and well, but a maternal grandfather and aunt had been insane, and in her mother's family several members are said to have been markedly eccentric. She has six brothers and sisters. One of the latter was for a time insane, her insanity caused, it is supposed, by a disappointment in love.

The present attack commenced about three or four months before her admission to the Hospital, with symptoms of irritability and violence. She had no illusions, delusions, nor hallucinations, but at times became so violent that she would throw china, or any article within her reach, at any person who happened to be near. Her insanity was preceded by depression of spirits; the lady for whom she worked often found her crying alone. Her menstruation has not been regularly established; during the last three years she has menstruated but two or three times, and then but scantily.

As this young woman's insanity began when she was between fifteen and sixteen years old, it comes within the period at which hebephrenia most frequently occurs. She has many of the silly peculiarities, much of the sham emotion, and much of the changeability in manifestations, which mark this form of mental trouble.

Some of the entries made from time to time in the case-book of the Hospital will indicate better than many words the peculiarities of the case.

"September 7th, 1886.—She displays as yet no violent tendency, but will tease and annoy other patients. She is clean and neat in her habits, and in good physical condition.

"September 28th, 1886.—The patient was transferred to another ward, because of difficulty with other patients in the ward in which she was first placed. After her transfer, she succeeded in making her escape, but was brought back before she had gone far. After her return she became very violent, breaking the window glass with her closed fist, spitting at the nurses, and using the most threatening and abusive language. She had to be secluded and restrained.

"October 15th, 1886.—Her physical condition is good, but there is much moral disturbance; she is untruthful, dishonest, and treacherous." On October 21st, she was quite amiable and easily managed for a few days during the week, but was again violent last night. On November 21st, it was noted that she was in good physical condition and was growing stouter and stronger. She had menstruated this month. She was much more obedient, was quite gentle, and was very fond of reading. In December it was also noted that she was quite well behaved this last month and had been taken home by her family to spend the Christmas holidays.

"January 20th, 1887.—The patient had not been home more than a few days before her sister found it necessary to return her to the asylum, because she was so violent and difficult to get along with.

"February 12th, 1887.—The patient is remarkably well behaved, is quite industrious and helpful about the ward, and seems happier than at any time since she has been in the house.

"March 8th, 1887.—She is excited and threatening in manner.

"April 23d, 1887.—She has just recovered from an attack during which she has been violent and sullen, and destructive.

"June 19th, 1887.—The patient is now quiet, well behaved, and gentle in disposition and quite industrious."

CASE XX.—*Hebephrenia in the Female followed by Secondary Dementia.*

Reported by Dr. Harriet Brooke, Assistant Physician.

E. C., aged 27, white, single, a domestic, was admitted to the Hospital January 29th, 1887. She was a tall, slender, stoop-shouldered woman, with brown hair, blue eyes, clear complexion, and a not very intelligent expression. Her pulse and temperature were normal, her tongue clean, bowels constipated, and menses regular. Examination of the urine gave a negative result.

(This woman's insanity began twelve years ago, when she was between fifteen and sixteen years old, and it is for this reason that her case is considered here; she appears to be a case of hebephrenia, or the insanity of pubescence, which has now passed into mild secondary dementia, a not infrequent result.)

At times, since the beginning of her insanity, she has been

quite excitable, and even violent, and has often wandered from home. Her father is still living; her mother is dead, and she claims that her step-mother has treated her very unkindly. Charges of this kind, however, are frequent both among the insane and the sane, and all such accusations must be carefully investigated before they are accepted, when made by the insane.

The following are a few extracts from the Hospital records:

February 7th, 1887.—Her mind is feeble, but she is clean in her habits, industrious, and good-natured.

February 14th.—She shows no change from the previous week, except an increasing obstinacy that was not apparent during the first week.

February 21st.—She appears about the same, no change since the last record.

April 10th.—The patient cries at night without any apparent cause and in a hysterical manner. Her physical state is good.

May 24th.—The patient is about the same as last month. She continues to cry at night. Chloral is sometimes given her at bedtime, and occasionally a hypodermic of hyosine.

June 19th.—Her physical condition is good. She sometimes cries in the daytime without any apparent cause, and in a few minutes will be found laughing immoderately at nothing.

Studying her mental condition, I find some dementia, but not advanced.

#### CASE XXI.—*Hebephrenia in the Male.*

Reported by Dr. Allen J. Smith, Assistant Physician.

H. M—, aged 18 years, white, single, born in Philadelphia, was admitted to the hospital December 22d, 1886. His parents are still living, and he has no family history of insanity or of any of the hereditary diseases.

Six months previous to his admission, he had an attack of intermittent fever. He denies venereal taint. He is not addicted to alcoholics, but smokes and chews to some extent. He admits masturbation, the practice extending over a number of years; his sexual organs are poorly developed.

On admission to the hospital, he was in a condition of apparently profound melancholia, with delusions of sinfulness. He would wander about and mutter: "God help me!" He made a pretense of searching for a knife with which to kill himself, but published the fact of his search about the ward. On two or three occasions, he has made ridiculous pretensions of committing suicide, but never going about it in a way that could possibly terminate fatally. He is quite bright and, if need be, very active; he has several times escaped from the institution by quite ingenious methods. Depression, while existing to a certain degree, is by no means so profound as would be supposed from first appearances, being readily cast off for a light-heartedness that seems also largely assumed. In speaking of his delusion of sin, he says "the condition

in which he is is the sin." For the most part, however, he must be closely questioned before he confesses to anything of this sort, and there is the possibility of it being assumed for a purpose. He will not look one in the face, and bears all over him the appearance of a masturbator. Since his admission, he has improved much physically as well as mentally. His mind, generally speaking, acted rather slowly on admission, but his memory was good. At present he is certainly brighter and more cheerful than before; but at times he shows an incoherence that used never to be noticed, and its existence is against a favorable prognosis.

The following is a report of a conversation between Dr. Mills and the patient:

Q. What are you chewing?

A. Chewing tobacco.

Q. You heard what the doctor said about you; are you insane?

A. Certainly I am.

Q. Are you much worried?

A. No, sir.

Q. Do you now think that you committed the unpardonable sin?

A. God knows whether I did or not.

Q. Do you think that you have?

A. I could not tell you.

Q. Do you sleep well?

A. Yes, sir.

Q. Were you ever in love?

A. No, sir, I work hard every day.

Q. Don't you think that hard work and being in love are consistent?

A. I could not tell.

Dr. Mills remarked that this case measured well up to the type of hebephrenia. One thing against this idea was the apparent existence of the definite delusion of having committed the unpardonable sin. The person who held such a delusion was more likely to be a true delusional melancholiac. The countenance of this patient was not that of a patient with melancholia. His delusions, if real, had not the solidity of melancholia. He exhibited a general silliness of behavior, and on several occasions had made pseudo-attempts at suicide. As a rule, these patients did not make genuine attempts at suicide. He also showed a tendency towards secondary dementia, rather than towards recovery.

#### CASE XXII.—*Hebephrenia in the Male.*

Reported by Dr. Allen J. Smith, Assistant Physician.

L. C—, aged 19, single, native of Philadelphia, broom-maker, with common-school education, was admitted to the hospital January 15th, 1885. He is a fair-complexioned, blue-eyed youth,

of medium build and delicate constitution. His family history shows some hereditary taint, a grand-uncle having died insane. His mother died of phthisis. He is an illegitimate child, and it is thought that brooding over his mother's wrongs had something to do with his mental disturbance. For several years he was regarded as queer and altered, but the active attack of insanity was quite recent at the time of admission. His eyesight failed him when a boy, and for many years he has been almost totally blind; he is unable to read any but the raised print of a few books intended for the use of the sightless. He has marked and constant nystagmus.

Throughout the course of his case, there has been constantly a religious tinge to his insanity. On admission, he proclaimed himself Melchizedek, and announced himself a chosen one of the Lord. This religious state has continued, with varying degree, all along, and still exists; and during the intervening time he has imagined himself various religious characters, as Nebuchadnezzar and Jesus Christ.

A specimen of his talk is as follows: "I am Nebuchadnezzar, the chosen prophet of the Lord. He speaks to me in thunder and out of clouds.—I should like to be a train master, standing in the depot at Harrisburg calling out, 'All aboard to Pittsburg and points in the West.'—I should like to cohabit with prostitutes, and become the father of illegitimate orphans."

Shortly after admission, he attempted suicide by some drug which he supposed was laudanum, but, fortunately, it was some harmless preparation. He is recorded as having made a marked improvement several months after admission to the hospital, but this was temporary. He is at present in much the same condition as at first, showing, however, some mental deterioration, a growing slowness in thought, and more incoherence. His delusions of personal identity are not so strong as described on admission.

The following report of a conversation between Dr. Mills and this youth serves very well to indicate the general mental condition of the patient:

Q. What is the matter, L——?

A. Nothing serious, except the Bible is a nuisance.

Q. What else?

A. It is a nuisance. They quote the Bible in the School for the Blind.

Q. Were you in the School for the Blind?

A. I was there. They are severe, but not intentionally; but it is open to question by mistake.

Q. I believe you said you were the Prophet Jeremiah?

A. I would rather be Jesus Christ.

Q. Why?

A. Because He is a healthier person.

A case of imbecility, J. C——, aged 16, was shown by Dr. Mills, as a contrast to the cases of hebephrenia, with the following remarks:



"This case can be dismissed in a few words. It is of most interest contrasted with the other cases. He comes to us for consideration at the present time, simply from the accident that he was admitted about the age of puberty. From his history, he has evidently been in this low mental condition since birth or infancy. The term imbecility is a proper one if used in a proper way. It is a mental disorder which can be traced back to birth. Practically, it is often nothing more than a high grade of idiocy."

Conversation between Dr. Mills and the patient :

Q. How old are you ?

A. Three months.

Q. Where do you live ?

A. Home.

Q. Where is your home ?

A. Fourth street.

Q. What is this place ?

A. Hospital.

Q. Who is this gentleman ? (Indicating Dr. Smith.)

A. Dr. Weeks. (A former resident.)

In examining such a patient, we must test his memory, his judgment, his knowledge of surroundings, etc. It is sometimes highly important to decide whether a person is or is not an imbecile, and, in the second place, if an imbecile, of what grade of intelligence. In such a case, the patient should be tested by making him write, by asking him with reference to the value of money, etc., as follows :

Q. (To patient.) How much money are you worth ? A. Three dollars.

Q. What are you going to do with your money ? A. Buy something.

Q. Are you going to get married. A. Yes.

Q. Who are you going to marry ? A. Beau.

Q. Who is your beau ? A. Mary.

Q. Mary who ? A. Mary Coe.

Q. Mary Coe ? A. Coe.

Q. Again ? A. Again.

Q. And again ? A. Again. (Echo speech.)

## Periscope.

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### ANATOMY OF THE NERVOUS SYSTEM.

**The Trigemini Roots.** W. BECHTEREW. (*Neurol. Centralbl.*, 1887, p. 289.)

The ascending root of the trigeminus arises from cells which extend in a column from the level of the pyramidal decussation up to the point of exit of the root from the pons. The lowest part of this column is continuous with the posterior horn of the spinal cord. The fibres arising from the cells of this column turn laterally and pass upward in a bundle, semilunar shaped in area, which increases in size as it ascends. This root of the fifth nerve develops later than the other roots in the foetus. The descending root of the trigeminus joins the ascending root; their fibres mingle and make their exit together. This root has no connection with the motor nucleus.

The motor root of the fifth arises in part from the so-called motor nucleus, in part from fibres which can be traced in the raphé, and thence, after decussating with the opposite root, into the motor nucleus of the opposite side. B. denies that there is any direct connection between the trigeminus nerve and the cerebellum. These results are derived wholly from a study of foetal brains.

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**The Hypoglossal Nuclei.** P. D. KOCH. (Rev. in *Neurol. Centralbl.*, 1887, p. 291.)

The author has studied the hypoglossal nuclei in a number of the lower animals and in man. He finds that the hypoglossal roots have no connection with the olivary bodies. These roots arise chiefly from the hypoglossal nuclei on the floor of the ventricle. Each nucleus is divisible into an outer group of large cells and an inner group of smaller cells. The nucleus is covered on its posterior surface by a thick layer of nerve fibres which run parallel with the axis of the medulla, and are seen in sagittal sections to connect the cells of the nucleus lying at different levels. Koch names these the *fibræ propriae* of the nucleus. There are other fibres mingled with these which join the two nuclei with one another. A few of the fibres of the hypoglossal roots arise from the anterior or accessory nucleus in the *formatio reticularis*. This nucleus is the representative in the medulla of

the anterior horn in the spinal cord—a fact clearly seen in pigeons.

Fibres which pass from these nuclei into the raphé and so join the anterior pyramids of the medulla are considered the means of union of the nuclei with the cortical tracts. M. A. S.

#### PHYSIOLOGY OF THE NERVOUS SYSTEM.

##### **The Heat Centre.** (*Gazetta Degli Ospitali*, 17th Aug., 1887.)

PROF. GIRARD recently reported upon this subject before the Società Eleveziana Delle Scienze Naturali the result of his experiments upon rabbits. He has come to the conclusion that the cerebral centre of thermogenesis is in the corpora striata. Each lesion of the median portion produced an increase of heat well marked, which was not the result of spasm of the vaso-constrictor nerves of the skin. Exciting this region with electricity, there followed a notable increase of heat, justifying the assertion that increase of heat is the result of excitation and not of paralysis. Moreover, after the puncture and excitation of this region of the brain there was considerable increase of urea in the urine, indicating an increase of combustion in the organism; this was accompanied by a perceptible emaciation of the animal.

Girard localized the centre of heat in the median portion of the corpora striata and down toward the base of the brain, and affirms that this apparatus increases the heat under excitation and notably influenced and regulated the production of heat. He thinks that artificial increase of heat is not identical with that of fever. Increased production, at the same time diminished dispersion, of heat from the body are, according to his view, the two conditions essential to fever. GRACE PECKHAM.

##### **The Situation of the Emotional Centres.** K. PONTOPPIDAN. (*Centralbl. f. Nervenheilk.*, 1887, p. 521.)

There are certain organic brain lesions in which emotional manifestations such as laughing or crying appear without cause; or in which an emotional cause produces undue effects, *e. g.*, a pain produces laughter. Such symptoms are usually associated with symptoms of disease of the pons and medulla. Recent investigations show that the centres affected in such cases are those in the vicinity of the vasomotor centre in the pons. The author describes three such cases. In the first, any question caused the patient to laugh. In the second, laughing or crying occurred indiscriminately when any attempt at conversation was made by the patient. In the third, fits of laughter occurred without apparent cause—the mere entrance of any one into the room would produce one. In two of these cases autopsies showed the existence of apoplectic clots in the crura cerebri and pons Varolii, and other symptoms of pons disease were present.

Is it not more probable that emotional manifestations are simple reflex acts, and that their occurrence on slight cause may indicate not a diseased "emotional centre," but a disturbance in the inhibitory tracts which control the reflex centres? M. A. S.

**The Concentration of the Blood as a Condition of Stimulus for the Central Nervous System.** (*Lo Sperimentale*, May, 1887.)

At the Royal Physiological Laboratory at Florence DR. IRO NOVI made some interesting experiments upon animals to show the effects of concentration of the blood on the nervous system. To do this he injected into the jugular veins of dogs a ten per-cent solution of chloride of sodium, withdrawing from the carotid a similar amount of normal blood. When the amount of chloride of sodium thus added was equivalent to double that found in the blood normally, convulsions occurred in all the muscles of the body.

2. In similar conditions, it did not change hæmoglobin into methæmoglobin. The action in this respect was different from that demonstrated by Marchand for the alkaline chlorates.

3. The peripheral nervous apparatus, muscles and nerves, are not influenced by this condition of the blood, and in the central nervous system, neither the spinal cord nor the medulla, but only the brain is affected.

4. The loss of water which the brain has undergone, especially the gray substance, is the cause of the musculo-nervous system attaining to such a high pitch of excitation.

5. In all probability it is the same cause which produces the convulsions which accompany an analogous but pathological concentration of the blood, as for example in Asiatic cholera.

GRACE PECKHAM.

**PATHOLOGY OF THE NERVOUS SYSTEM.**

**The Brain of the Deaf and Dumb.** J. WALDSCHMIDT. (*Allg. Zeitsch f. Psych.*, xliii., 4.)

Waldschmidt describes the brains of two deaf and dumb individuals. In both the operculum, inferior frontal convolution, temporal lobe, and island of Reil were imperfectly developed, and the deficiency was more noticeable in the left hemisphere. The island of Reil in both was noticeable on account of the lack of convolutions, its thinness, and the rudimentary appearance of its anterior portion, and these peculiarities were more marked on the left side. The author believes that such a deficiency in the island of Reil, by impairing the power of conduction of impulses between the temporal and frontal lobes, is sufficient to cause the condition of deafness and dumbness.

The subject needs further observations.

**Chronic Progressive Ophthalmoplegia Externa, with a Description of a Special Group of Cells in the Oculo-motor Nucleus.** C. WESTPHAL. (*Arch. f. Psych.*, xviii., 846.)

This case is interesting because the number of autopsies so far made in ophthalmoplegia externa is very limited. The patient, a man 44 years of age, began to suffer from attacks of unconsciousness in 1881, which became quite frequent, each lasting several days. These were followed by mental symptoms of a hypochondriacal nature which caused his removal to an asylum. On admission, the existence of total loss of power in all the external eye muscles with divergent strabismus was discovered. There was double ptosis; the pupils did not react to light, but acted in accommodation. There was beginning atrophy of the left half of the tongue, paresis of the uvula, and evidence of locomotor ataxia. He died two years after admission, the symptoms persisting until death. The autopsy showed a degeneration of both third nerves, atrophy of the third nerve roots, so that no normal fibres were to be found in the crus in their position, and marked changes in the oculo-motor nuclei in their entire length. There was a want of cells in the nuclei, and the cells which were present were small, round, and without branches. But dorsad from the atrophic oculo-motor nuclei, and opposite their anterior portion, two groups of cells were found on each side of the raphé—one group lying near the median line (the median group), the other lying outside of the first (the lateral group). These groups were not affected by the atrophy. Westphal finds no description of these groups in the works on anatomy, and thinks that they have escaped observation on account of their position, which is so far anterior as to be near the opening of the aqueduct of Sylvius into the third ventricle. He is inclined to consider that the function of these groups is to control the acts of accommodation and of pupil reflex to light, and he explains the lack of reaction to light in this case by the existence of a spinal affection. The abducens nuclei were also atrophied in this case. The left hypoglossal nucleus was atrophic, and the sclerosis of the posterior columns of the cord diagnosticated during life was found.

The article closes with a discussion of recent observations on ophthalmoplegia externa, and is accompanied by drawings of the new groups of cells.

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**Syringomyelitis.** R. WICHMANN. (*Neurol. Centralbl.*, 1887, p. 343.)

The author has collected 33 cases of syringomyelitis—22 males, 11 females. The average age was 33 to 34 years. The average duration 3 to 4 years (from 2 to 7 years). In 6 cases the entire cord was involved. In 27 cases only a part of the cord, the lower cervical and upper dorsal regions being most frequently affected. As to the cross area involved, it was found that the posterior columns were very often invaded, the posterior horns less

frequently, the anterior horns rarely. In 17 cases, the cord had a tumor-like swelling at the point affected. In 11 of these cases, the disease extended beyond the boundaries of this swelling. The cavity in the cord varied greatly in size. As to the symptoms there was much diversity. In a few cases they were wanting. In the large majority severe pain was present, greater in the extremities than in the body. Partial anæsthesia was present in 17 cases—the loss of pain sense being especially noticeable—in one case being general, in one case being so complete that fracture of the bones occurred without pain. The paralysis is the most constant symptom—usually of the legs, often of the arms, sometimes of both. Disturbance in the genito-urinary and rectal mechanisms is frequent. The sudden change and alternation of symptoms, rapid improvement and then relapse, is quite characteristic of central glioma, as of all intramedullary tumors. As to the therapeutics, the only comment is in regard to hot baths which were found to hasten the progress of the affection. M. A. S.

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#### THERAPEUTICS OF THE NERVOUS SYSTEM.

**On the use of Galvanism in the Treatment of certain forms of Insanity.** By JOSEPH WIGGLESWORTH, M.D., Lond., Rainhill Asylum. (*Journal of Mental Sc.*, Oct., 1887.)

The author reports upon his experiences with the use of the galvanic battery in the treatment of insanity. By way of preface, he makes some very simple and elementary remarks upon the use of the galvanic battery. From his mode of application, we should say that it is not only important for *him* to use a galvanometer, but a good rheostat would be of good service, for he has used currents as strong as 25 (!) milliamperes, applying the cathode to the forehead and the anode to the nape of the neck. We do not marvel that one patient resisted a good deal as the strength (of the current) was increased, or that one patient, suffering from melancholic stupor, developed a wild maniacal attack under this treatment; nor is it surprising that others "disliked the applications immensely." If the author wishes to study the effects of galvanism upon the insane, let him use milder currents, and we think he will obtain the same if not better results. The author concludes (1) "that whilst the use of galvanism to the head is a proceeding which is certainly *not* going to revolutionize the treatment of insanity, this agent is nevertheless one that is capable of doing much good in certain selected cases, and that, by its judicious (*sic*) employment, we may every now and then cure cases which would otherwise drift into hopeless chronicity. (2) The class of cases which offers the best field for the employment of this agent is that which includes examples of mental stupor and torpor cases, which are grouped under the specific designations of *melancholia attonita* and so-called 'acute dementia.'"

B. S.

**Zur Toxicologie des Paraldehyd.** Prof. Dr. EUGEN FÖHNER. (*Berl. Med. Woch.*, Sept. 12th, 1887.)

The author has experimented with paraldehyde on animals (horses in particular), and has found that the drug is not as innocent a one as it is claimed to be. The continued use of this drug produces not only a condition resembling the state of chronic alcoholism, but brings about very marked changes in the structure of the blood-corpuscles. It is a powerful oxidizing and reducing agent. A horse was given a large dose of paraldehyde, not sufficient to produce hypnotic condition, and yet the blood-corpuscles had undergone extreme changes. In carnivorous animals, if the toxic effects had been attained, a condition resembling pernicious anæmia was established. The author fears that a similar condition might result from the long-continued exhibition of the drug in man. This we doubt, for we have seen it used for months without such effects, and furthermore, the hypnotic effects of the drug upon the human body are obtained with relatively small quantities, long before the toxic effects would be reached.

**On the Treatment of Neuralgias by Kataphoric Action.** (*Deutsche Med. Wochenschr.*, No. 39, 1887.) By Prof. ADAMKIEWICZ, in Krakow.

At the medical conference recently held, Prof. A. returned to the subject of the treatment of neuralgias by means of his diffusion electrode. A. claims that nothing will heighten the efficacy of a therapeutic agent as combining it with an agent of similar properties. The anode having the power of reducing the excitability of nerves, that action will be reinforced if a substance like chloroform be introduced into the skin under the action of the anode. It is now proved beyond a doubt that the chloroform does penetrate into the skin and parts below, but not very far beneath the skin. The author, therefore, recommends these applications in cases of neuralgias of superficial nerves rather than in case of sciatica for example.

Several cases are cited of severe supraorbital neuralgia which were relieved at once by the application of the anode taken with chloroform over the painful point, increasing the current gradually up to 7 MA. The method is well worth a trial, particularly in acute cases.

B. S.

**On the Prognosis and Treatment of Locomotor Ataxia.** M. BENEDIKT. (*Wiener Med. Presse*, 1887, Nos. 33 u. 34.)

Benedikt divides the cases of locomotor ataxia into several categories whose prognosis differs widely. The first group, in which the prognosis is most favorable, consists of the cases which begin with atrophy of the optic nerve. This particular symptom is, it is true, incurable, but the course of the disease is slow and extreme motor symptoms rarely develop.

The second group is made up of the cases with prodromal gastric crises. In these the prognosis as to the development of motor symptoms is not so absolutely favorable as in the first group, but the motor symptoms subside in two-thirds of the cases.

The third group contains the cases of dementia paralytica ascendens, with complicating tabes. In this group it is the rule that the spinal symptoms are limited to the loss of pupil and tendon reflexes and to the development of the Romberg symptom (swaying when standing with eyes closed). That it is not a matter of indifference that the ataxia does not go on, even though the cerebral process progresses, is evident, since the care of such patients is much easier when the spinal symptoms subside. (May these be cerebral ataxia?)

A fourth group with relatively favorable prognosis is made up of the cases in which the symptoms develop rapidly within a few weeks and soon reach a high pitch of intensity. These cases are often maltreated and hence do not recover. Antisyphilitic treatment, galvanism, and hydrotherapeutic measures are here of no use. The patients should have absolute rest with the most antiphlogistic treatment. (May these be cases of neuritis?)

A fifth group with a fairly favorable prognosis consists of tabetic patients in whom syphilis is the manifest cause of the disease. An atypical course of the case will awaken suspicion of syphilis, but the only criterion is the result of an antisyphilitic course of treatment. Such a course must be carefully conducted, routine inunctions being avoided. It may be stated that Benedikt does not accept syphilis as a universal factor in the causation of tabes.

The sixth group, whose prognosis is unfavorable, is made up of the cases which present the typical picture of the disease, though even a few of these may recover.

As to treatment, the author recommends strongly galvanism and hydrotherapy, giving, however, no sufficient details regarding his methods of applying these agents. He admits that great patience is needed and that treatment must be kept up for a long while. In the hyperæmic stage which is present in the rapidly advancing cases, ice to the back, absolute rest, wet cups, and the internal use of ergot and of nitrate of silver are the means recommended. In the syphilitic cases, hypodermatic use of corrosive sublimate is advocated. He considers nerve stretching as one of the most important means of treatment (a means, however, which has not found favor with any other neurologist of note and has been abandoned everywhere, excepting in Benedikt's clinic).

While it seems improbable that in any case where sclerotic processes have occurred in the spinal cord a true recovery from the symptoms of sclerosis can follow, it must not be forgotten that two such cases have been recorded by so good an authority as Schultze, of Heidelberg, in which, though the symptoms of tabes disappeared, subsequent post-mortem examination showed the



persistence of the lesion. These cases can only be explained upon the supposition of a vicarious action in the nervous system, the rôle of one set of fibres and cells being undertaken by another set.

M. A. S.

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**A Case of Tetanus Cured by the Hypodermic Injection of Cocaine.** By LOPEZ. (*El Genio Med. Quir.*, 1887. Quoted in *Gaz. Degli Ospitali* June 5, 1887.)

M. G., 50 years old, having worked in the cold and wet, complained of rheumatic pains in the back and extremities. Three days after he had an attack of opisthotonus and painful spasms and all the symptoms of idiopathic tetanus. Morphine and chloral hydrate were prescribed. For three days the patient, under the influence of these medicines, had little pain, but there were increased muscular rigidity and spasms. At last he was unable to swallow, and death was believed imminent. Injections of morphine were without effect. Then the writer injected three syringe-fuls of a mixture of morphine and cocaine, 5% of each. The effect was immediate. After two hours he could move the extremities, open his mouth and turn himself in his bed. The next day he continued to improve. There remained a slight trismus and a little rigidity of the neck. A quarter of a syringe-ful of the same solution was injected in each side of the neck. The day after, all the symptoms had disappeared and in a few days the patient gained strength and was able to return to work.

GRACE PECKHAM.

## Society Proceedings.

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### NEW YORK NEUROLOGICAL SOCIETY.

*Meeting of October 4th, 1887.*

*The President, DR. C. L. DANA, in the Chair.*

#### THE ARTHROPATHY OF TABES.

DR. W. H. PORTER presented a specimen of the spinal cord and the knee joint from a case of tabes. The patient was a woman, 32 years old. The family history was good. There was no history of alcoholism or rheumatism in the case. In May of the present year, ovariectomy had been done, and, with the exception of the formation of a ventral hernia, the patient had made a good recovery. The affection of the spinal cord dated from five months previously, and in February last a gradual increase in the size of the knee had been noted. As early as December 15th, however, the patient had suffered pain, and partial luxation had made standing difficult. Examination showed the right knee painful, but not swollen. The left knee presented subluxation, some fluid, and enlargement of the lower portion of the femur. There was some dyspnœa, also some headache. The patient also suffered with external hæmorrhoids, but the urine was normal, and the co-ordination good. The right knee measured 18 inches, and the left 21½. The patient was a large woman, weighing 300 pounds. The enlargement and the riding of the patella upon the joint were all the positive symptoms in the case, and the patient was transferred to the surgical ward. There continued to be a great deal of boring pain in the knee, but no puffy feeling in the feet, no anæsthesia, and no ocular trouble. The joint was excised with an apparently good result, but three weeks later, on August 10th, discoloration of the skin of the buttocks and of the small of the back appeared, and in the course of a day or two the part sloughed. The woman died, apparently of sepsis from the slough. Post mortem the viscera were found healthy with the exception

of the liver, which was pock-marked with cicatricial depressions. The right knee joint was found in good condition. Moderate changes of interstitial thickening were found in the spinal cord. The question was, whether this should be considered a case of ataxia, the only positive symptom being the boring pain. The supposition of tuberculosis had been entertained, but the excised joint had failed to show tubercular tissue or bacilli, and erosion of the bones had been the only discoverable change.

The PRESIDENT added that the spinal cord was now being examined by Dr. Græme M. Hammond, and there was no doubt that the posterior columns, particularly the postero-external columns, or columns of Burdach, were affected. It was a case of sclerosis of the cord, most marked in the posterior columns.

DR. W. A. HAMMOND suggested that articular affections with locomotor ataxia were infrequent in this country as compared with France. He had never seen a case, although many cases of locomotor ataxia had passed under his notice. According to Charcot, they were very common, resembling in this the *grande hystérie*, which also seemed limited to France, and even to the walls of the Salpêtrière Hospital.

DR. PORTER stated that during the past year he had made post-mortem examinations in four cases of locomotor ataxia. In two of these there were joint affections, and in two there were none. They had been recognized during life in only one of the cases. He had never seen the condition before this year.

DR. L. WEBER referred to a case which had been presented the day before at the Society of German Physicians. He had seen others, but not such as would answer to Charcot's descriptions. Rosenthal, of Vienna, also had described severe joint affections in locomotor ataxia, but not in such great numbers. The speaker could point to a few cases out of 70 or 75, but as a rule the affection had not been destructive. It was acute; it would come and go, and complete restitution might occur. He referred to two cases. In one there were diabetes and locomotor ataxia, and in the other there was sclerosis of the cervical region of the cord. In this case it looked as if the cartilage and bone were invaded, as there were crackling and change in form. A case like that referred to as presented the day before, with intra-capsular fracture and such extreme mobility, he had never seen before in this country.

The PRESIDENT, from an experience embracing two cases, had

found that the term tabetic arthropathy did not mean the same thing under all conditions. One of the patients, whose case was of ten years' standing, slipped and fell, nearly dislocating a joint already loose, so as to present a typical arthropathy of this disease.

MELANCHOLIA DUE TO THE PROLONGED USE OF MORPHINE.

DR. S. B. LYON presented the history of a case of melancholia following the prolonged use of morphine for the relief of cardiac pain.<sup>1</sup>

DR. M. PUTNAM JACOBI considered the case very interesting. The conception of the ego as a simple unit was childish. Undoubtedly changes might occur in the groupings which went to form the consciousness of which the ego was made up. In this case there was destruction of the ordinary linkings of consciousness, with a replacement of the ordinary normal impressions by the formation of a new sphere. The aberrations produced by morphine and other toxic influences could be explained in like manner. The suddenness of the recovery in this case further demonstrated that the forced paths of association had ceased to be travelled and the old ones been resumed—a process which might be likened to the switching of an engine from one track to another.

DR. HAMMOND objected to the term double consciousness in connection with the case. Double consciousness might be represented by plus and minus. The patient was not at the same moment in both conditions, but at different periods led separate lives, during the plus condition knowing nothing of what occurred during the minus condition, and *vice versa*.

DR. E. C. SPITZKA thought that the reader of the paper had used the term with the meaning of the French. The case was assuredly not one of double consciousness as the term was used by alienists. In double consciousness there was really a double ego with alternate consciousness.

DR. LYON recognized that the case was not one of double consciousness as Dr. Hammond and Dr. Spitzka had used the term. The patient had, however, a double consciousness in an ordinary acceptance of the word, with a real and an unreal aspect, the latter dominating.

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<sup>1</sup> See this number.

A DISCUSSION ON THE USE OF ANTIPYRINE AND ANTIFEBRINE IN  
NERVOUS DISEASES.

was opened by DR. T. S. ROBERTSON. He had used antipyrine first, in general practice, in acute rheumatism, and it had appeared not only to reduce the fever, but to control the pain. This had led him to try it in a case of migraine which had resisted aconitine and chloral. He had subsequently given it in a hundred unselected cases. In 90 per cent of these, relief had been obtained in from fifteen to forty-five minutes. He had given it without a stimulant, and diaphoresis was the only ill effect which he had seen. In the severe pains of tabes he had given from ten to twenty grains hypodermically, or from twenty to thirty by the mouth, with a resulting diminution in frequency. In insomnia, the sleep was better than that produced by chloral. The speaker had never seen heart failure. In hyperpyrexia he gave forty grains at a dose, and had himself taken one hundred and twenty grains in the course of twenty-four hours without any unpleasant result.

DR. E. WAITZFELDER had had an experience somewhat similar to Dr. Robertson's, but had given the drug with a stimulant, having found nausea and vomiting common where this was not used. He had given antipyrine in twenty miscellaneous cases of headache, producing relief in about fifty per cent. He had directed its use in the epileptic ward about three months ago. It had been thoroughly tried, but without any appreciable effect. He had used it for the pains of locomotor ataxia, but, while the patients had improved, he did not attribute this to the drug.

DR. HAMMOND had used antipyrine, and his experience had been entirely negative. He had given both antipyrine and antifebrine, singly and combined, in neuralgia, the pain of locomotor ataxia, insomnia, vertigo, and headaches both of the anæmic and of the hyperæmic variety. His method of administration had been to give fifteen-grain doses three times a day, continued for from two to three weeks. A new drug was not needed to shorten an attack of migraine. A hypodermic injection of morphine would do it, or one hundred grains of bromide of sodium, or  $\frac{1}{10}$  of a grain of nitro-glycerin, according to the variety. In a case of tuberculous meningitis in a child of two years, he had given four-grain doses of antipyrine for the relief of pain, and had signally failed. He had given it in epilepsy without result.

The PRESIDENT asked whether Dr. Hammond had given antifebrine in epilepsy.

DR. HAMMOND had given both antipyrine and antifebrine in fifteen-grain doses, with similar results, sometimes combining seven grains and a half of each. He referred to the insolubility of antipyrine, making its administration difficult.

DR. G. W. JACOBY thought it serviceable to hear the other side of this question. His own experience had resembled that of Dr. Hammond. Antipyrine gave some relief in migraine, at least following the first or second administration. It sometimes cut short insomnia, but that about comprised its usefulness in this field. It was not without danger. He had seen collapse from a dose of twenty grains.

DR. M. PUTNAM JACOBI had given antifebrine in the infirmary to a child with pleuro-pneumonia, and, with lowering of the temperature, relief from pain also was produced, although the physical signs remained unaltered and defervescence occurred in a typical manner on the sixth day.

DR. SACHS believed that antipyrine could be recommended only in migraine, possibly also in headache of a neurasthenic type. Cases should be followed up for a number of months. The results from two or three administrations should not be relied upon. He had given it in a dozen cases of migraine, with relief within twenty minutes after its first administration, and in no case had it been necessary to repeat the dose more than two or three times, at intervals of an hour. The only unsatisfactory cases had been those of the spastic type. The paralytic type had been in every case relieved. In one case, that of a man of thirty-two years, the condition had resisted all previous treatment. The patient every four weeks had to go to bed for from twelve to thirty-six hours, and was incapacitated for work for several days. Antipyrine did not entirely relieve the headache in this case, but the patient was able to continue his business during the paroxysm. In the speaker's experience, about 20 per cent of the headaches of neurasthenic origin were relieved. In headaches of anæmic and gastric origin, the treatment had been unsatisfactory. In insomnia, with and without migraine, antipyrine had appeared to act as a true narcotic. Sleep of nine hours and a half to ten hours followed the administration of two grains. He had used it in the lightning pains of locomotor ataxia and in peripheral neuralgias, particularly trigeminal and sciatic, without result

Antipyrine was not a panacea, and the speaker thought that it should be used carefully. He had met with no bad results himself, but from the reports of others it was evident that such results could occur.

The PRESIDENT expressed surprise that Dr. Hammond had been so unsuccessful in the use of antipyrine, and suggested that he keep a closer watch upon his clinical assistants. Dr. C. H. Brown professed to have been cured of a most violent migraine by antipyrin, and was enthusiastically prescribing it in Dr. Hammond's clinic. In the treatment of epilepsy, antipyrine could only be used empirically. Antifebrine, on the contrary, had been shown to act as a spinal depressant, and hence could be rationally prescribed.

DR. ROBERTSON had found antipyrine soluble in Vichy water. He considered a dose of from twenty to thirty grains safe. He had not maintained that it cured, but that it acted as a palliative in the conditions named.

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*Meeting held Tuesday evening, November 1st, 1887.*

*The President, DR. C. L. DANA, in the Chair.*

DR. BEVERLY ROBINSON presented a case of

#### APHASIA WITHOUT PARALYSIS.

The history had been furnished by the assistant house physician to Charity Hospital. The patient was 68 years of age, born in this country and admitted to Charity Hospital Aug. 25th of the present year. His previous history was negative in regard to syphilis. He once had had rheumatism in the knees, but the date was not known. His present difficulty dated from May, when he commenced to have headache. Subsequently he fell out of bed to the floor. He was able to crawl back into bed, but from this time his speech was affected. He was treated in Bellevue Hospital, and was from there transferred to Charity Hospital upon the date named. Upon entry he was apparently in perfect physical health excepting speech. Objectively there were no signs of paralysis. His walk was slow but good. Possibly the right leg dragged a little. The dynamometer registered twenty with the right hand and with the left hand ten. The faradic reactions were normal in the upper extremities. In the lower extremities they were somewhat quicker and stronger upon the left than upon

the right side. The sight in the right eye was as good as before the injury. The right eye was found more hypermetropic than the left, but the optic disks showed the physiological cupping in both eyes. The urine was negative. When admitted the patient could speak only in monosyllables, using most frequently the phrases "Yes," "No," "That's it," and "Exactly." He read the newspaper and apparently understood what he read. He also used gestures and explanatory words. When asked his age, as "Are you 40?" he would answer "No;" "32?" "No;" "48?" "No;" "68?" "Yes." If given a pencil and paper and told to write his age he would put down an 8 and in front of it a 6, adding "That's it." He could not write his name, writing John in the place of Isaac. It thus appeared to the speaker a case of amnesic and ataxic aphasia with agraphia, depending upon lesion of the foot of the left third frontal convolution. His own interest in it had been directed to the question whether lesion of this area would involve the intrinsic muscles of the larynx. On account of the difficulty of obtaining intelligent co-operation, however, a satisfactory examination could not be made. The treatment had consisted in the administration of 15 grains of pot. iodid. t. i. d.

DR. DANA asked whether the patient could copy and whether mirror writing was obtained with the left hand.

DR. ROBINSON did not think that the patient could copy, but would test the point.

DR. DANA suggested that his own name be not used, and the President's was substituted. The patient wrote Charles Dane. Dr. Jacoby added another phrase and found that the patient wrote *s* for *m* and *f* for *i*.

DR. STARR would rule out amnesia in the case. The terms amnesic and ataxic aphasia had respectively a sensory and motor significance. Here the patient apparently understood perfectly, and his difficulty was purely ataxic or motor.

DR. ROBINSON explained that a few weeks ago the patient had not understood well, and that even at the present time he would probably not understand all questions which might be put to him. Yet, during the last two or three weeks, he had very markedly improved.

DR. BALL asked how carefully the question of sensory ability had been tested. When told to do certain things, would he do them? Also, would he do the same when written directions were given him?



DR. ROBINSON replied that the patient had complied with oral directions ; that written directions had not been tried.

DR. DANA asked whether the patient was able to utter exclamatory language. In some cases where ordinary speech was lost through lesion of the left third frontal convolution, profane or ejaculatory language was still obtained through the corresponding centre upon the right side.

DR. ROBINSON stated that the patient did not present this peculiarity.

DR. PUTNAM JACOBI asked whether the test suggested by Dr. Ball could not then be made.

DR. BALL directed the patient to take hold of Dr. Robinson's left thumb. The man hesitated and apparently failed to understand, but complied when directed simply to "Take his thumb."

DR. STARR asked the patient whether he read the papers and understood them, and the man replied, "Yes, of course."

DR. BALL mentioned the case of Dr. Chas. Allen, aphasic for several years and now dead. This case had, during most of the time, read and apparently understood, yet it was probable that he obtained the sense from leading words, and that a large number of the words he did not understand at all. Probably the same was true of this case.

DR. ROBINSON asked whether there was any recognized difficulty in the use of the intrinsic muscles of the larynx for phonation associated with the aphasic condition.

DR. STARR had been interested in this subject through a paper by Dr. Delavan in regard to a cortical centre for the larynx. For a year he had sent to Dr. Delavan all cases of hemiplegia coming to him at the Polyclinic and the Demilt Dispensary. Fifteen or sixteen cases had been examined without the discovery of any affection on either side of the larynx. He believed that no such case was upon record. It was certainly contrary to the general experience of neurologists to find any difficulty of phonation connected with aphasia. In fact, Dr. Ross, in his last edition, had stated that in the lesion of aphasia the muscles of the larynx were not affected.

DR. ROBINSON had retained an impression that the literature showed such cases. He had had his attention directed to the subject in this way.

DR. STARR asked whether a patient could have this paralysis of the larynx without being hoarse.

DR. ROBINSON replied that he could. He had seen patients with partial paralysis of a vocal chord who were not hoarse.

DR. GRAY stated that a distinction should be made between hemiplegias from lesion of the internal capsule, and those from lesions of other parts, particularly the pons and the medulla. Of six or seven cases in which hemiplegia was due to hemorrhage, embolus, or thrombus in the internal capsule, he had had careful examinations made by competent laryngologists, and in none of them was there any paralysis of the laryngeal muscles. In hemiplegia from other causes, alteration of the tone of voice was sometimes obtained. In true and simple aphasia he thought that the voice was not affected. Dr. Dana added that in pseudobulbar paralysis the larynx was involved, the lesion being in the corpus striatum.

DR. H. C. COE followed with a paper upon

#### THE SIGNIFICANCE OF PELVIC PAIN.

Pain was not a reliable indication of disease. Often an epithelioma of the cervix would cause less distress than a dislocation. The description of pain by the patient and the localization of its cause by the physician represented separate topics for thought. As described by the patient the pains of the pelvic regions were, in general terms, an aching pain in the lower part of the sacrum, a shooting pain in the inguinal regions, and the gnawing pain of carcinoma. All of these pains would be referred to some lesion of the peritoneal or connective tissue, or both—to some plastic exudation not necessarily of great amount. The distress caused by a retroflexed uterus was much greater where there were adhesions than when there were not. It was fair to assume that this constant aching pain was due to the implication of nerves in the exudate. Laceration of the cervix, excepting that extending into the vaginal fornix, did not, in itself, cause pain. The cervix was a very insensitive organ, and laceration was but a link in the chain of circumstances which resulted in pain. Malignant disease even did not necessarily give rise to pain. Hart and Barbour say that there is no pain so long as the cervix is affected, Hewitt says that the pain of cancer is due to localized attacks of peritonitis. The pain was earliest and most severe when the growth was in the body, thus differing from sarcoma of the body, in which there was little pain. Possibly in this variety of cancer the intra-muscular nerves were involved in the growth. The shooting,

darting, sickening pains associated with disease of the tubes was due to nothing but peritonitis. Hegar refers to cicatricial nodules in the broad ligaments, and even in the case of ovarian neuralgia it seemed probable that the pain was due to pressure upon the nerve before it entered the organ rather than to changes within it. Otherwise this pain would not be relieved by the relief of perimetrial adhesion, as frequently occurred.

The inference was to give a guarded prognosis in regard to the relief of pelvic pain. If the pain, associated with a fissured cervix, was due to cicatricial nodules in the broad ligament, we might cure the laceration and the endometritis, and yet the pain would continue. To remove the ovaries for the relief of pain was even more hazardous.

The speaker thought that gynecologists exaggerated the frequency of reflex pain. With Dr. Dana, he considered anæmia the most frequent cause of vertex headache. Pelvic reflexes were found in the upper lumbar and intercostal nerves. He had not found sciatica of ovarian origin, according to Dr. Mundé's suggestion. It might occur as the result of some exudates, but must be rare as a reflex pain. Dr. Mundé himself somewhat oddly remarks that this pain is relieved by a blister over the sciatic notch. Dr. Polk's plan of separating adhesions for the relief of pain presented scarcely less risk than the usual operations referred to. Treatment by electricity according to the methods of Apostoli gave the most satisfactory results. Reflex or transferred pains might also be due to inflammatory foci, and might be treated in the same way.

DR. GRAY, as a neurologist, felt at a loss to know how to discuss such a paper. Many pains besides those of pelvic origin centred in the back, such as muscular pains and the pains of peripheral neuritis. He had himself often referred patients to competent gynecologists for examination, and had found nothing in the pelvis to account for pain over the sacral, lumbar, or coccygeal vertebræ. Yet, on the other hand, one could not deny the capricious vagaries which distinguish the truly reflex pelvic pain.

DR. PUTNAM JACOBI remarked that while specialism assisted investigation, it was a misfortune to the patient. She did not consider that the writer of the paper had proved his position. Pelvic pain might be more definitely mapped out. The uterus, the central organ of the pelvis, was supplied by the lumbar

plexus; and lesions of this organ were accompanied by pain in the track of the lumbar nerves. Two or three other definite points were known. Pain referred to the distribution of the femoro-cutaneous nerve was the most characteristic accompaniment, not of ovaritis, but of ovarian neuralgia. Again, pain in the end of the spine might be spinal, ovarian, or endometrial in origin. A retroverted uterus without peri-uterine lesion would cause aching in the sacral region, but no coccydynia. The speaker considered pain from pelvic exudation rare and somewhat hypothetical. Even chronic peritonitis gave only a dull, aching pain which was quite tolerable except when the patient was moving about. The worst case which she had ever seen, one which finally died from an exacerbation, was comfortable when in bed. The ganglion had been found frequently diseased in cases of pelvic pain, especially in those associated with posterior perimetritis, and in some cases of violent hysteria, it had been found atrophied. This ganglion, situated between the body and the cervix, was often the site of excessive tenderness, and a permanent neuralgia might result from a perimetritis which would persist long after the removal of its cause.

DR. RANNEY quoted Dr. Beard as having said that the nervous system is like a mountainous region, in which echoes are returned with equal intensity from distant parts. This description was peculiarly applicable to pelvic pain. As a general practitioner he had frequently treated pelvic pain locally, without relief, in cases where it had finally disappeared upon the removal of a distant cause. It was not, in his opinion, possible to establish the seat of any pain unless removal of the supposed cause had established cure. He had failed to find compliance with this formula in the interesting paper under discussion.

DR. DANA stated that he had been disappointed often when sending patients with pelvic pain to gynecologists for examination; and that painful neuroses, even, had in some cases resulted from gynecological treatment.

DR. PECKHAM considered that the reader of the paper had underrated the suffering from direct pressure as a factor of pelvic pain. A retroflexed body or the hypertrophied cervix of an anteflexed body might cause a good deal of suffering by direct pressure upon the sacral nerves. Again, pain in the right or left iliac region might often be attributed to tension where, with a shortened broad ligament and lateral deviation of the uterus,

there was pulling upon the ligament of the opposite side. Pain persisting after operation might later be found to have disappeared. The eye which looks on the sun retains for a time its image. Thus the nerves of other parts after prolonged irritation retain the impression of that irritation and the habit of pain after the removal of the cause. Time is thus required in these cases to perfect a cure.

The speaker indorsed the value attributed to electricity by the author of the paper. Whether it acted by direct influence upon the nerves themselves or by modifying the pelvic circulation was not apparent.

DR. STARR read a preliminary report of the Stevens Commission.

The meeting adjourned.

## PHILADELPHIA NEUROLOGICAL SOCIETY.

*Stated Meeting, October 24th, 1887.*

MORRIS J. LEWIS, M.D., *in the Chair.*

DR. J. T. ESKRIDGE presented a paper on  
MENTAL AND NERVOUS DISEASES AS INFLUENCED BY THE CLIMATE  
OF COLORADO.<sup>1</sup>

DR. F. X. DERCUM reported  
A CASE OF UNILATERAL CONVULSIONS AND ANOTHER OF HEMI-  
PLEGIA OCCURRING IN URÆMIA.

The following cases are of interest in connection with the observations of Raymond,<sup>2</sup> and of Chantemesse and Tenneson,<sup>3</sup> in the same field, and also in connection with two cases of hemichorea associated with Bright's disease, recently reported by the writer.<sup>4</sup> They are as follows:

*Case I.*—H. S., colored, aged 69 years, an inmate of the Philadelphia Almshouse, had, while in the out wards, occasionally suffered from convulsions. On several occasions he had been admitted into the epileptic ward of the hospital. Here it was noticed that he usually had several convulsions in close succession, each lasting about five minutes, and that after the attack was over he would fall asleep. His convulsions were always general, and attended with frothing at the mouth.

Mentally he was much impaired. He had delusions of persecution and sometimes imagined himself unable to walk. He was

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<sup>1</sup> See this volume, p. 554.

<sup>2</sup> Raymond: "Sur la pathogénie de certains accidents paralytiques observés chez des vieillards, leur rapports probables avec l'urémie," *Révue de Médecine*, Sept., 1885.

<sup>3</sup> Chantemesse et Tenneson: "De l'hémiphrégie et de l'épilepsie partielle urémiques," *Révue de Médecine*, Nov., 1885.

<sup>4</sup> F. X. Dercum: "Two cases of Hemichorea Associated with Bright's Disease," *JOURNAL OF NERVOUS AND MENTAL DISEASE*, xiv., August, 1887.

also at times violent and maniacal during the night, requiring to be strapped in bed.

On August 15th, 1887, being much improved, he was, at his own request, again transferred to the out wards.

On August 27th, after having had occasional fits for several days, he was again admitted to the epileptic ward. On this day his convulsions seemed much worse than usual. They were almost continuous, and he was brought into the ward in a semi-conscious condition. In striking contrast with the seizures previously observed, the spasms now existed only on the left side, beginning apparently on the left side of the face, extending quickly to the left arm and thence to the left leg, being less severe in the last-mentioned member. During the quiescent periods it was observed that the left arm was paralyzed, as was also to some extent the left leg. The left side of the face was also paralyzed, the mouth was drawn toward the right, and the left cheek was flaccid. The conjunctiva of the right side was sensitive, but that of the left was not. The urine was drawn and examined for albumin, but none detected. The temperature was  $97.2^{\circ}$ ; pulse 140. The sphincters were relaxed.

After the convulsions had ceased altogether, the patient slept. Consciousness had evidently been absent during the attack, but, on the following morning, August 28th, it had fully returned. The condition of left hemiplegia, however, persisted. His urine was now examined a second time, and revealed both albumin and casts, the latter in large numbers.

On the evening of the 28th, the convulsions recurred, the patient having nineteen in rapid succession. On the morning of the 29th, he was again conscious, and talked, but during the day he gradually became weaker and died at one o'clock.

An autopsy was held within the following twenty-four hours, and revealed the following: Calvarium of moderate thickness. Dura very adherent, the brain being removed in the calvarium. Pia not thickened, but its meshes very œdematous. Veins of vertex full. Pia presented milky opacities here and there, and was easily separated from the convolutions, a sub-pial space having been formed, which was distended with lymph; vessels of base extremely atheromatous. Brain as a whole soft, flattening by its own weight. Ventricles large and excessively pale. Choroid plexuses very pale and cystic. Velum interpositum presented whitish granulations. On section, the cortex was found pale,

and the white matter excessively œdematous. Serial section of both hemispheres *failed absolutely* to reveal a *focal lesion*.

In the general post-mortem nothing of special importance was found except that the *kidneys were markedly granular* and fatty. The lungs were emphysematous, the heart fatty, and the aorta somewhat atheromatous.

*Case II.*—J. H., aged 82 years, white, in the out wards of the Philadelphia Hospital, complained of weakness in the left arm and leg. This continued for four weeks, when he was admitted to the nervous ward of Dr. Mills, temporarily under my care. Here it was noticed that he dragged the left leg a little in walking and that he moved his left arm with difficulty. Sensation on the affected side was also dulled. The hemiparesis grew gradually more and more pronounced and was as sharply defined as a hemiplegia of organic origin, which, indeed, it was at first supposed to be. At last, though sluggish mentally, he was able to reply to questions and to make his wants known in his native language, namely German. Gradually, however, he failed in mind and body, incontinence of urine, delirium, and comæ set in, and after having been in the ward two weeks he died. During life the urine had been examined and revealed a small amount of albumin.

On the day following death, the autopsy was held and the following conditions noted. Calvarium and dura normal. Pia arachnoid loose and very œdematous, its meshes milky. Vessels of base very atheromatous. Ventricles excessively pale. Choroid plexuses presented numerous large cysts. Velum interpositum infiltrated. Brain very soft. On section, found to be œdematous throughout. Careful serial sections *revealed no focal lesion*.

The kidneys were found to be *much contracted and cystic*, markedly cirrhotic.

The other organs revealed nothing of consequence. The lungs presented some adhesions in the right pleura, the heart valves were thickened, the aorta was atheromatous.

Both of the above cases are doubtless to be relegated to affections of the nervous system occurring during and as a consequence of Bright's disease. Chantemesse and Tenneson<sup>1</sup> have already recorded two instances of unilateral epilepsy occurring in the course of this affection and have verified one of them by post-mortem examination. Their cases are in every way a counter-

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<sup>1</sup> Loc. cit.



part of the case here reported. The convulsions were strictly limited to one side, and the autopsy revealed merely general œdema of the brain and chronic interstitial nephritis.

The objection may be made that these phenomena of hemiplegia, hemichorea,<sup>1</sup> and hemi-eclampsia are due simply to atheroma of the arteries of the brain, by means of which a portion of the cortex or a tract of the internal capsule may receive an imperfect blood-supply, be shut off, as it were, from its necessary amount, an oligæmia and œdema being the result. The weight of evidence, however, is against this interpretation. There are absolutely no areas of local softening or local necrosis to be detected, and the œdema is not confined to a spot but is general, is evenly diffused, and apparently as much in one hemisphere as in the other. Besides, the ingenious experiments of Raymond<sup>2</sup> leave no doubt as to the possible one-sided action of the nervous system in uræmia. It will be remembered that he first mutilated the sympathetic system of an animal by removing the superior cervical ganglion of one side. In this way he threw the entire nervous apparatus, so to speak, out of balance. He then ligated the hili of both kidneys, and the convulsions that ensued were limited to one side. He repeated the experiment a second time with the same result. It seems as though the two halves of the nervous system bear unequal powers of resistance and that the weakest yields.

DR. OSLER asked if, at the autopsy, special attention was paid to comparing the two sides of the brain. It was some time since he read the articles to which reference had been made, but his impression was that more œdema had been found on the side of the brain opposite to the side on which the paralysis was present. An interesting point in these cases is that it appears to be absolutely impossible in certain cases to make a satisfactory diagnosis between cases of hemiplegia from apoplexy and cases of hemiplegia from uræmia. He recently went over this question with his class in the case of a man brought into the hospital with hemiplegia, unconscious, and with albumin and tube-casts in the urine. He said that it was in all probability a case of hemorrhage, but that it was impossible to exclude positively hemiplegia due to uræmia. He said that it might be a case of œdema of the brain from uræmia, in which the œdema, being more marked on one side of the brain, or from some other cause, the paralysis was uni-

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<sup>1</sup> Dercum, loc. cit.

<sup>2</sup> Loc. cit.

lateral. He had seen one case of unilateral convulsions occurring in a man who was brought into the General Hospital at Montreal some eight or nine years ago. He had convulsions for three or four days. At the autopsy in that case nothing was found in the brain that would account for the convulsions. At that time we were not so familiar with œdema of the brain, or with the relation of œdema of the brain in Bright's disease, as we are now.

DR. JAMES HENDRIE LLOYD said that Dr. Dercum may recall the fact that, at the meeting of the American Neurological Society two years ago, he referred to a case similar to those reported. The patient was a boy, aged seventeen years. When he saw him he was in a comatose condition with convulsions, which were confined to the right side of the body. The boy had been a chronic sot, and died a few hours after he saw him. At the autopsy there was nothing found in the brain to account for the condition. The only lesion was the "pig-back" kidney. This case was undoubtedly one in which the symptoms resulted from uræmia. He thought that in the intervals between the convulsions it was possible to demonstrate the fact that the side on which the convulsions occurred was paralyzed.

DR. MORRIS J. LEWIS said that a man, aged twenty years, a heavy drinker, under his care, had albumin with granular and oily casts in his urine. He suffered with frequent convulsions, which usually followed the periods of heaviest drinking; and on two occasions the convulsions were most marked on the right side, and were followed by paresis, which lasted two or three days. The young man was placed in a position where he could not obtain alcohol, and has apparently made a complete recovery. There is at present no albumin, no casts, and no paresis.

DR. DERCUM said that, to the best of his recollection, the authors to whom he had referred, have noted no local œdema. He had made three post-mortems in cases of well-marked hemi-disease occurring during uræmia, and he had found no more œdema on one side than on the other. The cases reported by Drs. Lloyd and Lewis are of special interest as occurring in young persons. Most of the cases reported have occurred in old people. With reference to local cerebral manifestations from a general cause like uræmia, he recalled the case of a gentleman under his care for several years who suffers from chronic contracted kidney. He now and then has general uræmic convulsions. Some years ago he had the following remarkable seizure. He went down town to

attend to some business, and while in a bank he suddenly became aphasic. He was perfectly conscious. The aphasia continued for several hours. This looks like another instance of a local manifestation from a general cause.

MENTAL AFFECTIONS ASSOCIATED WITH CHRONIC BRIGHT'S  
DISEASE.

DR. WILLIAM OSLER said that, as it was in close connection with the paper of Dr. Dercum, he should like to make some reference to the occurrence of certain mental affections which come on in connection with chronic Bright's disease. It is well known that certain mental phenomena occur in connection with chronic renal diseases besides simple uræmic coma. He had reported one case of violent mania in a man aged forty-two years, the subject of Bright's disease. When brought to the hospital, he had been maniacal for three or four days. He subsequently became comatose and died. A very interesting case was recently under his care in the University Hospital. A man was brought to the hospital Thursday evening. He saw him on Saturday. He was then quiet, in a semi-dozing condition, but could be aroused and gave a very intelligent account of himself. The whole clinical picture was that of chronic interstitial nephritis. He thought it not improbable that the man might pass into a condition of coma. There was nothing to attract special attention to his mental condition, and he did not regard his condition as critical. That night he got out of bed in the absence of the attendant, wandered about the ward, and finally jumped out of the window. It was subsequently learned that, before admission to the hospital, he had been violent, requiring two or three men to hold him. We were not told this when he was brought to the hospital. He had no doubt that this was an instance of mental disturbance due to chronic nephritis. He was told by one of the physicians who had attended him that the man was full of delusions. He thought that his wife and others were persecuting him.

He saw another interesting case a year ago last Christmas. This occurred in the practice of Dr. Mullin, of Hamilton, Canada. Here there was also a medico-legal question. It was whether or not the man was in a condition to make a will. There was no doubt as to the existence of chronic Bright's disease. The mental condition was peculiar. He believed that his wife and others had designs upon his life, and it was with difficulty that he could be

persuaded to take food. He thought that people were persecuting him. Although he gave a very intelligent account of himself, it was not considered advisable that he should make his will at that time. He was placed upon a somewhat more active treatment than he had previously received. This man subsequently did well, his mind had cleared, and he recovered sufficiently to get about and to make his will.

DR. DERGUM said that the negro who was the subject of unilateral convulsions had also presented, at various times, periods of maniacal excitement and had also had delusions of persecution.

## Editorial Notes and Miscellany.

THE Sept.-Oct. number contained the statement that Dr. Ott's article on the Thermogenetic Apparatus was published in the JOURNAL OF NERVOUS AND MENTAL DISEASE. The article in question appeared in the *Therapeutic Gazette* for August, 1887.

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THE Medico-Legal Society makes the following announcement :

The Medico-Legal Society of New York announces the following prizes for original essays on any subject within the domain of medical jurisprudence or forensic medicine :

1. For the best essay—One Hundred Dollars, to be known as the Elliott F. Shepard Prize.
2. For the second best essay—Seventy-five Dollars.
3. For the third best essay—Fifty Dollars.

The prizes to be awarded by a commission, to be named by the President of the Society, which will be hereafter announced.

Competition will be limited to active, honorary, and corresponding members of the Society at the time the award is made.

It is intended to make these prizes open to all students of forensic medicine throughout the world, as all competitors may apply for membership in the society, which now has active members in most of the American States, in Canada and, in many foreign countries.

All details of the award will be determined by the Executive Committee of the Medico-Legal Society of New York.

The papers must be sent to the President of the Medico-Legal Society of New York, on or before April 1st, 1888, or deposited in the Post Office where the competitor resides, on or before that day.

The name of the author of any paper will not be communicated to the Committee awarding the prizes.

All persons desiring to compete for these prizes will please forward their names and addresses to the President or Secretary of the Medico-Legal Society of New York.

In case the essay is written in a foreign tongue, it should be accompanied by a translation into the English language.

THE Council of the American Neurological Association has issued a circular containing the following notice :

In order that the coming meeting of our Association (Sept., 1888) may represent to the best advantage the combined work and thought of its members, the Council is making arrangements to present, at an early date, an outline of the programme to be followed.

Two important subjects have already been arranged for: (1) The relations of Renal Diseases to Diseases of the Nervous System, to be discussed by Drs. R. T. Edes, F. X. Dercum, J. J. Putnam, and E. C. Séguin, and (2) The Pathology of the Muscular Dystrophies, to be discussed by Drs. B. Sachs, P. C. Knapp, G. W. Jacoby, W. R. Birdsall, and C. K. Mills.

These gentlemen will speak from written notes, and it is urgently requested that any others wishing to speak will prepare themselves in a similar manner, and by notifying the Secretary, secure the privilege of being called upon to speak before the debate is thrown open to the meeting.

It seems to the Council desirable that the reading of original papers should not occupy more than thirty minutes (less if possible) and papers read in debate not more than ten minutes, except the opening papers.

If more papers are presented than can be read, the Council will give precedence to those which seem to them the most important and original, and those of which the Secretary is earliest notified. For this reason, as well as to insure good discussions, members are urged to signify their intention to read at as early a day as practicable.

The Constitution provides that the Secretary must be notified of all papers at least one month before the meeting, but it is hoped that the co-operation of the members will make it possible to issue a provisional programme much earlier than this, and any one signifying his intention to make a communication of real value may be assured that sufficient time will be secured, for him and for those who wish to speak upon the same subject, to be fully heard.

(Signed) J. J. PUTNAM, M.D., *President*,

106 Marlboro Street, Boston, Mass.

GRÆME M. HAMMOND, *Secretary*,

58 West 45th Street.

WITH this issue, the present Editor's connection with the JOURNAL ceases. He feels it incumbent upon himself to make some sort of explanation of this step to the many friends and contributors who have assisted him in his endeavors to make the JOURNAL the foremost journal of neurology in this country. The ordinary excuse that the Editor retires "from stress of professional duties" is not offered in this instance. While the Editor's time is amply filled with the duties of active practice, he would not have shunned the arduous task of editing the JOURNAL OF NERVOUS AND MENTAL DISEASE; but he could not make time both to edit the JOURNAL and to look after its business interests. For the latter task the inclination, too, was wanting.

The JOURNAL has in many ways been a severe drain upon the Editor's resources, but it is some satisfaction to know that during the past two years the standing of the JOURNAL has been a very high one; and that it has been a credit to American medical science. It is to be hoped that the readers of the JOURNAL will subscribe to this opinion.

The JOURNAL now passes into the hands of Dr. G. M. Hammond, of this city, who enters upon his new duties with the best wishes of the retiring Editor.























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